ABSTRACT

OF

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An Abstract of the Proceedings

OF THE

Association of Life Insurance Medical Directors of America

FORTY-EIGHTH ANNUAL MEETING

The Forty-eighth Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania, New York City, on October 28 and 29, 1937.

The following members, delegates and guests were present at some time during the sessions: Doctors J. W. Abbott, H. H. Amiral, H. B. Anderson, E. M. Armstrong, T. M. Armstrong, W. B. Aten, J. A. Avrack, G. H. Barber, N. J. Barker, W. B. Bartlett, J. T. J. Battle, E. W. Beckwith, J. E. Bee, M. B. Bender, R. W. Benton, F. M. Beresford, J. R. Biggs, C. C. Birchard, W. F. Blackford, H. E. Bogart, J. E. Boland, William Bolt, E. C. Bonnett, J. T. Bowman, W. M. Bradshaw, F. G. Brathwaite, A. W. Bromer, J. L. Brooks, G. R. Brow, C. T. Brown, H. B. Brown, B. C. Bullen, B. F. Byrd, E. J. Campbell, J. H. Campbell, F. H. Carber, P. E. Carlisle, V. S. Caviness, C. L. Christiernin, C. P. Clark, A. D. Cloyd, E. A. Colton, H. W. Cook, P. M. Cort, H. W. Crawford, J. H. Crawford, H. C. Cruikshank, R. M. Daley, A. H. Davison, A. C. DeGraff, H. D. Delamere, P. G. Denker, E. J. Dewees, E. G. Dewis, T. H. Dickson, Jr., E. S. Dillon, W. W. Dinsmore, L. I. Dublin, J. H. Dunkley, O. M. Eakins, J. L. Evans, F. C. Evers, W. G. Exton, J. G. Falconer, R. K. Farnham, H. H. Fellows, W. E. Ferguson, R. M. Filson, P. M. Foshay,

R. A. Fraser, E. M. Freeland, H. M. Frost, F. I. Ganot, E. E. Getman, J. M. Gilchrist, R. A. Goodell, H. W. Goos, A. S. Graham, Clarence Grigsby, F. L. Grosvenor, Llewellyn Hall, Frank Harnden, L. E. Hathaway, W. C. Hausheer, J. K. P. Hawks, W. D. Heaton, C. E. Herron, W. L. Hilliard, D. W. Hoare, D. C. Hoffman, C. O. Hollinger, Byam Hollings, J. C. Horan, J. H. Humphries, Ross Huston, W. G. Hutchinson, W. C. Huyler, A. S. Irving, J. G. Irving, C. B. Irwin, W. A. Jacquith, A. O. Jimenis, G. E. Kanouse, Foster Kennedy, F. W. Kenney, R. H. Kennicott, E. F. Kerby, H. B. Kidd, H. B. Kirkland, R. J. Kissock, E. R. H. Kurz, W. P. Lamb, I. C. Lawler, L. H. Lee, J. M. Livingston, G. C. Lockard, Carl Lovelace, H. C. McAlister, George McCreight, F. H. McCrudden, C. B. McCulloch, A. J. McGanity, L. F. MacKenzie, George McLean, H. E. McMahon, W. T. McNaughton, F. W. McSorley, H. L. Mann, W. L. Mann, L. K. Meredith, R. C. Montgomery, A. E. Murphy, J. R. Neal, Otto Neustatter, C. F. Nichols, J. B. Nichols, Eduard Novak, W. L. O'Connell, Herbert Old, M. I. Olsen, A. E. Parks, W. O. Pauli, J. E. Pollard, R. W. Pratt, W. W. Quinlan, J. H. Ready, W. P. Reed, P. V. Reinartz, W. A. Reiter, F. P. Righter, D. C. Roberts, J. A. Roberts, W. C. Roberts, A. J. Robinson, F. W. Rolph, R. L. Rowley, W. W. Rucks, S. C. Rumford, E. F. Russell, S. B. Scholz, Jr., B. T. Schwarz, Albert Seaton, Hall Shannon, H. L. Shapiro, J. T. Sheridan, Arthur L. Sherrill, D. M. Shewbrooks, R. L. Shields, E. E. Simmons, R. R. Simmons, J. L. Siner, D. W. Skeel, W. A. Smith, W. B. Smith, H. F. Starr, D. F. Steuart, I. R. Stidger, A. C. Stokes, A. R. Stone, E. V. Sweet, L. G. Sykes, B. C. Syverson, H. F. Taylor, W. E. Thornton, I. G. Towson, Joseph Travenick, Jr., H. B. Turner, H. E. Ungerleider, A. P. Upshur, R. C. Voss, W. R. Ward, R. L. Weaver, G. R. Welch, D. E. W. Wenstrand, C. F. S. Whitney, E. S. Williams, R. L. Willis, C. H. Willits, F. N. Wilson, M. C. Wilson, G. E. Woodford, A. W. Young, L. S. Ylvisaker, E. T. Yorke, Messrs. L. F. Baker, Arthur Hunter, G. C. Kingsley, H. H. Marks, J. H. Miller, J. P. Mills, J. V. Purcell, Pearce Shepherd and P. V. Wells.

Total attendance at all sessions, 213.

President Brown in the Chair.

Dr. Brown—On behalf of the Association of Life Insurance Medical Directors, I wish to extend to you gentlemen who are here as delegates of the Medical Section of the American Life Convention a hearty welcome and to assure you that we are pleased to have you with us. During the meeting, you are entitled to all the privileges extended to members. We encourage you to enter freely into the discussions. You have the right to vote on all questions. Each member of this Association, I am sure, will be glad to know you personally.

It is our custom at this time to be favored with an address by the Chairman of the Medical Section of the American Life Convention. Dr. Walter E. Thornton will speak to us.

Dr. Thornton—This is a happy prerogative of my office as Chairman of the Medical Section of the American Life Convention. As the spokesman of the delegation of that body to this meeting, it is my pleasant duty and my privilege to convey to you the felicitations of the Section and to assure you of our continuing respect and admiration.

Our respective organizations were called into being by different forces and have developed in somewhat different environments. However, our activities have been for the most part either parallel or complementary and necessarily so, for our interests and our general objectives have been identical. As a matter of fact our courses have drawn closer and closer together. Many of us owe allegiance to both organizations. Members of the Medical Section continue to become associates and members of the Medical Directors Association and vice versa. This is a healthy development to be encouraged.

In the meantime we yield to you as the senior body and gratefully acknowledge the leadership which is yours by virtue of the admirable use you have made of your wider opportunities. You have done much more in insurance medicine than have we but we have studied the lessons you have taught and perhaps have added some of our own. At any rate we are conscious of our

being and, privately at least, we are proud of our continuous and healthy, not to say vigorous, growth.

At our last annual meeting we were honored with the presence and the words of your envoy, President Chester Brown. We appreciated the courtesy. That occasion was held in June and at the Broadmoor, Colorado Springs, in the inspiring presence of the majestic Rockies.

It is my special privilege to extend to every member and every associate of this organization, to his wife and to his family, a personal and pressing invitation to our next meeting.

I had hoped to be able to announce the program at this meeting but our plans have been delayed by the untimely and tragic death of Dr. DeWitt Smith, the program chairman. However, these duties now have been intrusted to the capable hands of Dr. Maurice B. Bender, who will announce the details at a little later date.

We have chosen for the site of our meeting the magnificent Grove Park Inn at Asheville, North Carolina. The dates will be June 16, 17 and 18. There is no part of the country where the weather is more delightful at that time of year. The air is warm and balmy, and the parks and mountainsides are a paradise of rhododendron and laurel and azalea in the riot of full bloom.

The time and the place have been chosen quite as much for the wives and families as for ourselves and our professional guests. Committees are already planning for their enjoyment. Visits are arranged to famous Mount Pisgah; to that world wonder, the Vanderbilt estate; and to other points of outstanding interest.

For lesser beings whose souls do not expand so freely to beauty there are the justly noted golf courses of the district and tournament play for real prizes under the auspices of the Section. Of course, there is bridge, too, and other more or less innocent pastimes.

Above all it is an opportunity to meet the other fellow whom you may or may not really know. The atmosphere is informal and congenial and the environment perfect.

I think we are justified in anticipating an unusual program. It is our hope that all who can will come. You will find a warm and happy welcome awaiting you.

DR. BROWN—Thank you, Dr. Thornton. We are indeed appreciative of these words of greeting and friendship.

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DR. BROWN—Our By-laws prescribe an order of business for the Annual Meeting and it is for that reason that I take a few minutes of your time to deliver what is prescribed as the:

PRESIDENT'S ADDRESS

It seems appropriate, as we gather in annual meeting, to assume for a few minutes an attitude of introspection: that we may endeavor to look into ourselves; to consider our history; to examine the present state of our existence; and to express our hopes and ideas for the future. Our membership during the forty-eight years of organization may be said to have comprised four generations. The founders and charter members of the Association may be said to have been the first generation. Many of these, through the years of their experience, had struggled individually with the medical problems associated with life insurance. They knew that their work differed from that of clinical medicine and that the questions daily presented to them for solution required an experience at variance with that acquired at the bedside. They had a feeling of lonesomeness. The trail which they should follow had not yet been blazed. They felt the need of companionship, the necessity for an interchange of thoughts and experience, and they realized that progress in this new branch of medical activity could best be speeded by concerted action. And so they founded this Association, infusing it with their ambitions and their purposes, and gave it to us. And these ambitions and these purposes remain today the ambitions and the purposes of the Association.

The second generation may be considered to be those who became members in the decade and a half which followed. They gave us a life insurance medical literature; they placed insurance selection on a rational and scientific basis, and they pointed the way out of the chaos of individual impressionism to an orderly method for the conduct of an important branch of life insurance.

They encouraged friendship, they gave of themselves, they lent a helping hand, and there were few who were not better medical directors because of what they acquired through these contacts. They trained, they educated, they developed the oncoming generation, and their influence extended far beyond the limits of their own companies. An efficient, well-rounded and competent organization representing the larger portion of life insurance medicine in the United States and Canada was the legacy which they passed on to their successors.

The third generation may be considered to be those who inherited that legacy. We are they. And we too have our task. It is the assumption of the responsibility of maintaining the high standards which have been established, of preserving and advancing the ideals for which our predecessors struggled, and of sustaining a velocity of improvement proportionate to the improvement in life insurance and in all branches of medicine.

But it is the fourth generation to which I wish to appeal, the younger men, all those who have within the last few years joined our ranks, and those who have not been active in our deliberations or in the conduct of the Association. Much is to be done in the years to come. The science and the art of medical selection are still young. Much remains unknown. Changing circumstances, and advances in medicine and in the other sciences and arts present new problems, but they also provide the means for solutions and improvements. Work and study have been and will continue to be the open-sesame. In no circumstances can we more swiftly and in a more dependable way travel toward the solution of our problems than in the surroundings which constitute these meetings.

Another advantage which has been afforded by these meetings and by this Association is that of personal acquaintanceship and friendship. Our work and our standing in the general scheme of life insurance would have advanced with much less speed had it not been for the opportunity to know each other more intimately. Possibly this one fact is sufficient reason for our existence. This spirit of camaraderie is to be encouraged. Those with fewer acquaintances here and those of the succeeding generation will find

the other members willing and eager to advance that spirit. Here there is neither east nor west, neither north nor south, nor any international line. Here the relative size of the company from which a member comes is not a factor. The very essence of our constitution clearly indicates that this Association is an organization of individuals.

And so may we remain alive to our responsibilities. May we keep in loving and appreciative memory those who gave us this organization and those whose untiring work and loyal efforts have brought it to its present fortunate state. May we make, cherish and maintain lasting friendships among ourselves, which will support and aid us in our individual duties. And may we, in these efforts, be helpful in maintaining the high position of life insurance in the eyes of all men.

* * * * * *

Dr. Brown—In 1933, Dr. Brathwaite, of the Equitable, spoke to us concerning the selection of risks with glycosuria and gave us some interesting information relative to the mortality experience of that company in risks of this sort. Dr. Brathwaite, Dr. Beckwith and Mr. Mills of that company have prepared a paper for us, bringing their experience up to date. Dr. Beckwith will speak to us.

A FURTHER MORTALITY STUDY OF GLYCOSURIA AMONG ACCEPTED AND REJECTED RISKS

FREDERICK G. BRATHWAITE, M. D., EDGAR W. BECKWITH, M. D., AND JOHN P. MILLS

Equitable Life Assurance Society

At the 1933 meeting of this Association, a paper was presented by one of us entitled "Glycosuria: A Selective Formula and Illustrative Mortality Study." The material covered in the former paper consisted of an explanation of a formula on which the selective process was based, which formula attempted to take into account the double criterion of both blood and urinary findings where blood sugar time curves were obtained. In addition to this, there were presented the results of a mortality study of cases issued from 1925 to 1931 with exposures carried to the 1932 anniversaries. The material included 2,346 standard entrants and 2,263 substandard entrants. The basic table used was that representing the standard experience from 1920 to 1926 issues observed to the 1927 anniversaries of companies contributing to the Joint Occupational Study 1928.

In the current paper it is our purpose to present, in a slightly modified form, a continuation of the previous mortality study through four additional years; *viz.*, issues from 1925 to 1935 carried through 1936 anniversaries; and to examine the results produced by an application of the same selective formula.

In addition to this, we shall present the results of an investigation made in 1934, as to the survival or cause of death in a group of 2,175 individuals whose applications were declined or who failed to pay the first premium on policies that were offered, and in which group, surprisingly enough, we succeeded in obtaining answers to nearly 94% of our inquiries—an unusually high percentage in studies of this nature.

We recognize the fact that any discussion of the subject of glycosuria in these days of conflicting opinions on the part of

clinicians and insurance companies is beset with many pitfalls. One has only to examine briefly the literature of the past few years, or to read opinions about individual cases, of which we have upwards of one hundred expressed by clinicians of considerable eminence, to appreciate the fact that there still exists much variety of opinion and debatable ground.

The fact that glycosuria and hyperglycemia are frequently concomitant with serious circulatory disease seems to be of increasing importance, but a definite cause and effect relationship does not appear to be as yet clearly established and the various theories with regard to different endocrine disturbances in certain cases have their advocates. Opinions on the relationship between glycosuria and various blood sugar findings show considerable divergence, as is evidenced by the numerous terms in use; such as renal glycosuria, diabetes innocens, potential diabetes, true diabetes, etc., and difficulty is encountered in finding agreement as to the exact definition of these various classes. For these reasons it is felt that, while one must bear in mind the physiology and pathology of glucose metabolism as a background, it is more properly within the scope of this paper to limit the discussion to the results which have been obtained by a life insurance selective process covering both entrants and non-entrants, including in the latter class those rejected and those who did not accept the offers which were made.

Mortality Among Insured Glycosurics

From the Society's issues of 1925-1935 inclusive there were extracted all standard policies loaded +5 or more for glycosuria either on examination or in the past and all substandard policies which had glycosuria as their only impairment of importance. These were observed to their 1936 policy anniversaries, the expected claims being calculated on the basis of the Equitable Standard Table 1925-1934 issues.

The procedure in the present investigation involves three important departures from the methods used in the earlier studies. (See Volume XX Transactions—Association of Life Insurance Medical Directors). At that time we used the Basic Table 1920-1926 which appears in the Joint Occupation Study 1928 for the

calculation of the expected deaths. Since then we have prepared a mortality table from our own standard examined business, mentioned in the preceding paragraph, which represents very closely the mortality experience over the period of the present study and this table has, accordingly, formed the basis of our calculations of expected claims.

The earlier table represents a mortality which, on the business as a whole, is probably about 10% lighter than the later table, but this relationship does not hold for all ages. The Basic Table 1920-1926 shows a heavier mortality at the younger ages at issue and a considerably lighter mortality at the higher ages, particularly at the later durations for issue ages 35-59. The effect of this is that the mortality ratios of the present study are in general lower for the older ages at issue than they would have been if the expected deaths were calculated by the Basic Table 1920-1926 and higher for the younger ages. For all ages combined these ratios would be somewhat higher if the study had been conducted on the old basis.

Secondly, the present investigation is by policies rather than lives. When there is an unusually high proportion of duplicate policies among the death claims, the mortality ratio for a particular group will be greater by the policy method than by the life method, and conversely, if the proportion of duplicate policies is unusually low, the mortality will be correspondingly lighter. This factor is important since it has already been noted in our past studies among glycosurics that the relationship of policies to lives among the death claims was apparently abnormally high in the standard group and abnormally low in the substandard.

The third difference arises from the fact that the present study is confined among the substandard policies to cases where glycosuria was the only impairment of importance. Under previous studies all rated up cases with a sugar debit were included in the substandard material without regard to the presence of other factors that may have contributed to the substandard rating. The new procedure has the advantage of showing the extra mortality that may be attributed almost entirely to the glycosuria impairment alone, since the substandard group does not include lives with major impairments other than glycosuria.

The policies issued at standard rates numbered 5,217 and a summary of the mortality by issue age and duration is given in Table I.

TABLE I
Glycosuria—All Standard Issues
(5217 Entrants)

Policy Year	Issue Ages to 39 Exposed	Expected	Claims	Ratio	Issue Ages 40–49 Exposed	Expected	Claims	Ratio
1–2	4,768	9.37	10	107±22%	3,250	15.48	14	90±17%
3–5	5,123	14.51	8	55±18%	3,521	29.02	41	141±15%
6–8	3,006	10.77	22	204±29%	1,973	24.04	21	87±14%
9–11	997	4.75	4	84±31%	664	9.72	7	72±21%
All	13,894	39.40	44	112±11%	9,408	78.26	83	106± 8%

Policy Year	Issue Ages 50+ Exposed	Expected	Claims	Ratio	All Ages Exposed	Expected	Claims	Ratio
1–2	1,579	19.40	20	103±15%	9,597	44.25	44	99±10%
3–5	1,788	37.27	43	115±12%	10,432	80.80	92	114± 8%
6–8	1,008	29.89	27	90±12%	5,987	64.70	70	108± 9%
9–11	337	10.93	17	156±25%	1,998	25.40	28	110±14%
All	4,712	97.49	107	110± 7%	28,014	215.15	234	109± 5%

The various substandard classes of the Equitable are shown in the following table, which gives the mortality assumed in the letter classification.

P—130–135
Q—140–145
R—150–170
S—175–195
T—200–245
U—250–300

In Tables II, III, IV, and V summaries of the substandard material by presumed mortality groups, issue ages and durations are given.

TABLE II
Mortality Group 130–145% (P&Q)
(1014 Entrants)

By Issue Age

Issue Age	Exposed	Expected	Claims	Lives	Ratio $\left\{\frac{\text{Claims}}{\text{Expected}}\right\}$
to 39	2,029	5.38	7	(6)	130±33%
40–49	1,571	12.89	8	(5)*	62±19%
50+	1,086	21.83	25	(19)*	115±15%
All	4,686	40.10	40	(29)	100±11%

*One life appears in both of these groups.

TABLE II—Continued
Mortality Group 130–145% (P&Q)
(1014 Entrants)

By Duration

Policy Year	Exposed	Expected	Claims	Lives	Ratio {Claims / Expected}
1–2	1,803	9.25	9	(5)	97±22%
3–5	1,720	15.77	15	(11)	95±17%
6–11	1,163	15.08	16	(13)	106±18%
All	4,686	40.10	40	(29)	100±11%

TABLE III
Mortality Group 150–195% (R&S)
(1228 Entrants)

By Issue Age

Issue Age	Exposed	Expected	Claims	Lives	Ratio {Claims / Expected}
to 39	2,311	6.18	10	(7)	162±34%
40–49	1,854	14.87	17	(13)	114±18%
50+	1,131	21.57	40	(24)	185±20%
All	5,296	42.62	67	(44)	157±13%

By Duration

Policy Year	Exposed	Expected	Claims	Lives	Ratio {Claims / Expected}
1–2	2,109	9.79	13	(8)	133±25%
3–5	1,810	14.76	20	(15)	136±20%
6–11	1,377	18.07	34	(21)	188±22%
All	5,296	42.62	67	(44)	157±13%

TABLE IV
Mortality Group 200–300% (T&U)
(686 Entrants)

By Issue Age

Issue Age	Exposed	Expected	Claims	Lives	Ratio {Claims / Expected}
to 39	1,384	3.72	6	(5)	161±44%
40–49	1,008	8.35	9	(8)	108±24%
50+	482	8.36	13	(11)	156±29%
All	1,874	20.43	28	(24)	137±29%

By Duration

Policy Year	Exposed	Expected	Claims	Lives	Ratio {Claims / Expected}
1–2	1,158	5.19	6	(4)	116±31%
3–5	976	6.96	8	(8)	115±27%
6–11	740	8.28	14	(12)	169±30%
All	1,874	20.43	28	(24)	137±17%

TABLE V
All Substandard Combined
(2928 Entrants)

By Issue Age

Issue Age	Exposed	Expected	Claims	Lives	Ratio $\left\{\frac{\text{Claims}}{\text{Expected}}\right\}$
to 39	5,724	15.28	23	(18)	151±21%
40–49	4,433	36.11	34	(26)*	94±11%
50+	2,699	51.76	78	(54)*	151±11%
All	12,856	103.15	135	(97)	131± 8%

*One life appears in both of these groups.

By Duration

Policy Year	Exposed	Expected	Claims	Lives	Ratio $\left\{\frac{\text{Claims}}{\text{Expected}}\right\}$
1–2	5,070	24.23	28	(17)	116±15%
3–5	4,506	37.49	43	(34)	115±12%
6–8	2,379	28.74	41	(30)*	143±15%
9–11	901	12.69	23	(17)*	181±25%
All	12,856	103.15	135	(97)	131± 8%

*One life appears in both of these groups.

In the standard material the most significant features are the general agreement with the previous study, taking into account the change in the basis of calculating the expected claims, and the closeness with which the mortality ratios of practically all the subdivisions by age at issue and duration approximate the ratio for the group as a whole. In fact the most important aspect of the matter is the uniformity of the entire picture. With about three times as many exposure years as in the previously reported experience the total mortality has changed only slightly and the subgroups have tended to level off as the larger volume of material has minimized accidental fluctuations.

The mortality among those accepted at increased rates of premium is not so uniform. The elimination of policies with major impairments other than glycosuria has appreciably lowered the general mortality, but certain other features have become apparent. Why is the mortality lower in the two lowest of the substandard mortality groups than in the standard? Why is the mortality only 137% in the group presumed to have 200-300% mortality and 157% in the 150-195% group? Why is the mortality at issue ages 40-49 so low in comparison with older and younger

ages throughout the substandard material? Is the rise in mortality shown for the durations beyond five years apt to continue, and what will its ultimate effect be?

Some of these questions appear to be interrelated. The abnormally low mortality in the age group 40 to 49 at issue (94% as shown in Table V) will probably correct itself in time: the first five year durations show only 68% mortality, while beyond that point the figure is 129%.

The situation of claims exactly equalling the expected in the group presumed to show 130-145% mortality is linked with the preceding situation. The low mortality in the age group at issue 40-49 is the determining factor. If instead of 8 claims this group had shown 13 deaths for 101% mortality, the group as a whole would have been raised twelve points to 112% of the expected. That the mortality for that age group should continue below 100% for long is decidedly unlikely, for if it did, we must conclude that glycosuria at those ages is preferable to absence of glycosuria. No one of us would be apt to defend such an hypothesis.

The reversal of mortality ratios observed in the 150-195% group and in the 200-300% group, by which the group in which the higher mortality was presumed showed more favorable results than the other, is explicable when the relationship between lives and claims is observed in the two groups. The ratio of claims to lives in the 150-195% group is 1.52 while in the 200-300% group it is only 1.17. If the usual ratio of 1.33 observed in many of our mortality studies had been true in each of these groups, the mortality ratios would have been exactly reversed as follows:

	Observed Mortality	Modified Mortality
150–195% Mortality Group	157%	137%
200–300% " "	137%	157%

These ratios are more nearly what might have been anticipated, but they are still distinctly below the rates provided for by the premiums. In this connection we must consider the last of the series of questions we asked ourselves above: "Is the rise in mortality shown for the durations beyond five years apt to continue and what will its ultimate effect be?"

To the first half of the question the answer is probably "yes". It was to be expected that for this impairment the mortality would be a deferred one, a certain proportion of the lives becoming frankly diabetic and eventually dying from the disease or its complications. An analysis of the causes of death by duration shows this to be true:

Policy Years	Total Lives Dying	Deaths Due to Diabetes	Percentage Due to Diabetes
1–5	51	2	4%
6–8	30	3	10%
9–11	17	5	29%

The fatal effects of the disease may be postponed but not eliminated. And this brings us to the second half of the question "and what will its ultimate effect be?" In the long run it would appear as though the rise in mortality at the longer durations in these substandard cases would tend to raise the mortality in each of the groups to a level approaching that presumed by the extra premiums charged and that, by and large, equity will have been preserved between the policyholder and the company.

In observing the results obtained by the use of the underwriting formula described in the 1933 Transactions of the Association, it was noted that the mortality in the standard group was reasonably satisfactory; in the first two substandard groups was very favorable and comment has been made above as to the possible reason; and in the second two substandard groups was entirely so; but that the results produced in the two highest substandard groups (T & U) appeared bizarre. A possible mathematical explanation has been given above but the question was also studied from the standpoint of the use of the formula itself. The figures obtained might lead one to question the validity of the formula to some extent, especially with respect to the mechanism producing the heaviest ratings. Since the formula involved the use of the double criterion of urinary and blood findings, it was decided to examine all the cases in this class (T & U) individually.

There was a total of 686 cases, and of these a glucose blood sugar test had been done in 156. The experiment consisted in rerating each case, with a modified formula which included the fasting specimen urinary findings but excluded all subsequent urinary findings

during the test. This was done on the theory that possibly the urinary findings after the ingestion of glucose weighed too heavily in the computation and that they should therefore be discounted, thus allowing predominance to the blood findings, but still maintaining the double criterion because previous Insurance Records and other current urinary findings prior to the test would still be taken into account. The results obtained from this procedure were as follows:

Number	of	cases	rated	lower	90
"	"	"	"	higher	18
"	"	"	"	same	33
"	"	"	"	indeterminate	15

The indeterminate cases arose entirely from cases which were accorded ratings in the early years of the current underwriting practice (1925-26) at a time when the rules were not as strictly defined and the data sometimes incomplete.

The conclusion might be drawn from this experiment that the formula was slightly at fault in permitting urinary sugar findings to weigh too heavily in cases having a blood sugar determination; or that—taking the entire T & U class with and without blood sugar determination—the mortality ratings accorded the larger amounts of urinary sugar were too severe. We are not prepared to comment on the soundness of the first conclusion inasmuch as our experiment was confined only to the peculiar results in the T & U classes, and did not include all cases of glycosuria having had a blood sugar determination. However, in view of the fact that in the standard group and the second two substandard groups the actual mortality approximated with reasonable accuracy the expected, it would appear that the formula was not seriously at fault, and that the second or alternative conclusion is the more probable one, *viz:*—that the ratings accorded to urinary findings were proper ones, although perhaps a little severe, for accidental and occasional glycosuria up to 1.0% and for intermittent and constant glycosuria up to .5%; but that the ratings for intermittent and constant glycosuria above .5% were very probably heavier than necessary. Furthermore, it seems reasonable to conclude that the use of the formula has proven satisfactory in the selection of risks with this impairment, and that

there is justification for continuing to observe the double criterion of a blood sugar time curve and urinary findings rather than rely on either factor exclusively.

It is, of course, obvious that a mass of material is of necessity excluded from consideration in a study of glycosurics by virtue of the fact that a large number of cases considered by a life company are declined; and another number of cases, though offered insurance, do not appear on the register of that company through failure to pay the initial premium. Hence no exposure (proof of survival or death) for any period can be recorded in these two groups from the usual company files.

With the hope that valuable information as to survival or death might be obtained from the material thus excluded, we undertook an inquiry among those cases declined for insurance and those failing to pay initial insurance premiums.

We read in the literature much of end results. Life insurance statisticians are accustomed to consider death only as an end result, as obviously the real end is death. In contradistinction to the term "cure" it is a much more definite end result.

A facetious, but none the less true definition of the term "cure", is that applicable to one who after reaching the quiescent period in disease attains a normal expectation of life. This is not without practical value statistically from a clinical standpoint.

Clinically the end result may be a cure or the removal of a patient from observation from one cause or another, not necessarily death.

We should avoid being led into wrong interpretations by a study of the causes of death. We should strive to determine the total excess of deaths (if any) to be expected in the disease under scrutiny. Further, in the study of deaths (end results) we must subordinate the cause of death in the interpretation of mortality.

All deaths occurring in a given group under study should be taken into consideration rather than those presumed to be related to the disease because:

1. Differences of opinion arise as to what causes should be considered as relative.
2. The limits within which a mortality ratio in any given study may be relied upon to reflect the true mortality picture are

determined by the probable error of the mortality ratio of the study. Where the actual deaths are greater than the expected, the probable error tends to increase as the number of deaths decreases (a fact peculiarly evident in clinical research) thus increasing the uncertainty of the results.

3. As a mortality study comparing a special group to a standard mortality table subdivided by causes of death is not practicable, we must include all deaths from whatever cause in the data available.

Life companies have exhaustively studied the mortality on assured lives, as it is on that material that they base their selective efforts. They have definitely proved the value of selection and the wisdom of excluding certain risks in some impairments or charging an extra premium rate in others. For some reason or another they have made little research in the mortality of those cases they have declined, after the usual scrutiny had been made in the selective process.

They have known that in excluding the bad or poor risk they may have excluded moderate or good ones, inadvertently so to speak, and have been content to do so. In other words, their efforts are directed more to excluding the undesirable risk than to including the possible risk from the standpoint of medical selection. With the hope, however, that a study of unobserved (declined and substandard not taken policies) might be of value, as stated above, we undertook the task.

It was quite surprising to find in this follow-up study among these cases that we could obtain proof of survival or death, with comparative ease in such a high percentage of the cases.

Mortality in a Group of Sugar Risks Declined or Not Taken Out

Our records of glycosuria contain cards not only for the cases of glycosuria where the policy was paid for but also for not-taken and declined business. During the year 1934 an effort was made to trace the lives in these latter two classes in order to study their mortality. An attempt was made to trace all persons whose surnames began with K or an earlier letter of the alphabet in the file.

There were 384 lives on the Not Taken Out list of substandard glycosurics about whom evidence of death or survival was obtainable on all but 28. Among the 1,791 persons refused insurance primarily because of glycosuria or hyperglycemia or both, information was lacking in only 203 cases. This represents 92.7% success in the follow up among the Not Taken Outs and 88.7% among the rejected, or 89.8% for the entire group.

What assumptions to make regarding the 7.3% of the Not Taken Out lives and the 11.3% of the declined, which may be classified as lost, should first engage our attention, for they cannot be disregarded entirely even though superfically it would seem to be possible to do so merely by omitting them from the tabulations. A little reflection, however, reveals that if we do this and accept the mortality ratios resulting from the consideration of the cases successfully traced as representing the mortality of the entire group we have tacitly assumed that the mortality in the "lost" group is the same as in the group traced. That is a reasonable, if not the most reasonable, assumption to make. Our guess is that the mortality rates in the "lost" group are distinctly higher than in the remainder of the data, and for the purposes of this study we shall assume that they lie somewhere between those of the observed group and five times those rates. Since in the case of the declined lives this results in rates between 16 and 22 times that experienced by standard insurance lives, we are not assuming rates of mortality that could with any justice be called light.

A test of this assumption may be made on the Not Taken Out group. They show a mortality of 152% of the expected by the Joint Occupation Study Basic Table 1920-1926 when only the observed cases are included, there being 9.23 deaths expected and 14 actually recorded. If the mortality in the group of 28 "lost" cases is assumed to be five times that of the other Not Taken Outs the total mortality for the group rises to 188% of the expected. The average rating of the whole group of Not Taken Out cases presumed a mortality of about 200% of the standard; but as there is a selection upon the part of the insured involved in refusing the policy, we might expect the observed mortality to be less than that allowed for by the rating.

The tables given below are based upon the lives that were successfully traced insofar as the figures representing the exposures, deaths and expected deaths are concerned, but in the "Ratio of Actual to Expected" of the two percentages given, the first represents the results obtained by leaving out the "lost" cases (assuming their mortality to be identical with the traced ones), while the second assumes a mortality ratio in the "lost" group of five times that in the group traced. This assumption is applied to the mortality ratio of the entire age group at issue regardless of duration. For example: the issues to age 39 in the Not Taken Out class showed 162% of standard mortality for all durations combined and accordingly, the deaths assumed to occur in the "lost" group at those issue ages were taken to be 8 times the standard expected for each duration. These approximations were considered justifiable in view of the nature of the whole problem.

TABLE VI

Substandard Not-Taken-Out Policies from Glycosuria Investigation (1934)

Expected Deaths by Jt. Occ. Study Basic Table 1920–1926

(Lives)

Year Following Exam.	Issue Ages to 39 Exposure	Deaths	Expected	Ratio	Issue Ages 40–49 Exposure	Deaths	Expected	Ratio
1	177	1	.35		111	2	.40	
2	144	–	.36		104	–	.52	
3 & 4	216	–	.65		150	1	.95	
5–8	140	2	.49		79	3	.62	
Total	677	3	1.85	162%	444	6	2.49	241%
	Adjmt.	1.62	.20		Adjmt.	1.20	.10	
		4.62	2.05	225%		7.20	2.59	278%

Year Following Exam.	Issue Ages 50+ Exposure	Deaths	Expected	Ratio	All Ages Exposure	Deaths	Expected	Ratios Orig.	Adjusted
1	93	1	.86		381	4	1.61	248%	—277%
2	87	1	1.12		335	1	2.00	50%	— 93%
3 & 4	124	1	1.83		490	2	3.43	58%	—107%
5–8	57	2	1.08		276	7	2.19	320%	—340%
Total	361	5	4.89	102%	1482	14	9.23	152%	
	Adjmt.	1.78	.36		Adjmt.	4.60	.66		
		6.78	5.25	129%		18.60	9.89		188%

The adjustment for the 28 "lost" lives accordingly presumes that 4.6 of them are dead.

TABLE VII

Declined Applications from Glycosuria Investigation (1934)
Expected Deaths by Jt. Occ. Study Basic Table 1920–1926
(Lives)

Year Following Exam.	Ages to 39 Exposure	Deaths	Expected	Ratio	Ages 40–49 Exposure	Deaths	Expected	Ratio
1	408	7	.83		579	9	2.17	
2	331	3	.86		468	7	2.46	
3 & 4	455	3	1.38		640	12	4.17	
5–8	245	4	.83		275	10	2.12	
Total	1439	17	3.90	436%	1962	38	10.92	348%
	Adjmt. 12.00		.54		Adjmt. 21.33		1.25	
		29.00	4.44	653%		59.33	12.17	488%

Year Following Exam.	Ages 50+ Exposure	Deaths	Expected	Ratio	All Ages Exposure	Deaths	Expected	Ratios Orig.	Adjusted
1	683	31	6.82		1670	47	9.82	479%	—611%
2	572	22	7.61		1371	32	10.93	293%	—453%
3 & 4	732	32	11.20		1827	47	16.75	281%	—417%
5–8	327	20	6.19		847	34	9.14	372%	—418%
Total	2314	105	31.82	330%	5715	160	46.64	343%	
	Adjmt. 46.67		2.92		Adjmt. 80.00		4.71		
		151.67	34.74	437%		240.00	51.35		467%

The assumption among the declined cases gives rise to the presumption that 80 of the 203 "lost" lives are dead.

It is obvious that we have by no means exhausted the material available. Our inquiries were directed to the applicant, to persons likely to know the applicant, to other life companies known to have insured the subject prior to consideration by us, to the examining physician, to persons given as references in the original record, or to any other person likely to be aware of the subject's condition, or of his demise, and in rare instances by inquiring through inspection sources.

It was quite an easy matter to obtain information as to survival or death and though we made no special request for information as to the subject's condition, if living, we did inquire as to the cause of death, if deceased. Much gratuitous information was volunteered by informants in most cases and where deaths had occurred the cause was ascertained.

The object of these studies, especially as illustrated in Tables VI and VII, has to do with the study of glycosuria which is pre-

sumably of clinical importance. At a glance, therefore, it will be seen where, from the standpoint of the life insurance investigator, we begin our studies and where we leave them to be taken up by the clinician. Conclusions, which we hope to arrive at as time goes on are directed therefore, almost exclusively, to the search for evidence of insurability.

In this search we arrive accidentally, so to speak, at conclusions not entirely contemplated by the clinical study of this condition. As we search among those with glycosuria for evidence of insurability, which is less easy to obtain than evidence to the contrary, we collect a mass of data which reaches beyond our own field into that of clinicians, but at what point the transition occurs it is difficult to say. We do not, and cannot proceed beyond a certain point except to include data, if we have any, which gives us mortality experience; hence prognostic opinions.

Our thought is, that we ought not abruptly to discontinue our observations at a point where the category becomes obviously clinical; and, on the contrary, that the clinician should not confine his studies to the clinical epoch solely, but should take into consideration pre-clinical studies as well. This would seem to suggest a liaison more intimate than exists between one set of investigators and the other. Life insurance companies should be only too glad to coöperate in this respect if demands were made upon them for data which they are obviously so well equipped to contribute.

The deductions that may reasonably be drawn from an analysis of these tabulations when restricted to a consideration of mortality, seem to naturally fall into two divisions, diagnostic and prognostic, without reference to treatment or other clinical criteria. As such they represent simply studies of the course of untreated glycosuria in the pre-clinical stage with the object of classification and prognosis.

It is interesting to note that in the study of mortality among assured lives in certain abnormal conditions (Medical Impairment Study) published in 1929, the criteria considered in measuring mortality in glycosuria were confined solely to its incidence and the amount of urinary sugar content, criteria which at this time would be considered entirely inadequate. In the group of "not specified"

cases or in cases in which glycosuria was found in only one specimen, all issued at standard rates, with an exposure of 86,584 and a death ratio of 107±3, the death rate from diabetes was five and one-half times the normal. From the tabulation it is shown that notwithstanding the low mortality ratio as a whole, deaths from diabetes were abnormally high, from which it is indicated that a certain number of the group were at the time of entry either actual or potential diabetics, which incidentally might be true of any group. The fact remains, however, that groups of those having glycosuria die of diabetes in greater numbers than those who are not glycosuric, even though they may die at a rate but slightly in excess of the normal.

Comments on the Study of Non-Insured Lives

In Table VIII are shown the data obtained.

TABLE VIII

The following are statistical details:

1.	Number of inquiries made		2175
2.	Number of replies received		1954
3.	Percentage of 2 to 1		89.8%
4.	Exposure years	7197 years	
	Average	3.7 years	
5.	Total Dead	174	
6.	Age distribution	to 39 558	
		40–59 653	
		above 60 743	
			1954
7.	Causes of death: see schedule (Table IX)		

1. The surprisingly large amount of information gained shows that life companies have the opportunity, if they wish to avail themselves of it, of studying mortality not only among assured lives but also among those who, for one reason or another, have at one time been denied insurance; in other words, of observing both sides of the picture.

2. Unlike the usual studies of mortality made by insurance companies, any mortality analysis of "declined" or "not taken" business is likely to be misleading. Some lives properly included in the group of declined or not taken cases fail to be accounted for

at a subsequent date. To disregard these lives and base conclusions upon the remaining ones, that have been accounted for, would not be satisfactory. We may not assume that the rate of mortality among the lives "unaccounted for" is necessarily the same as among those known to be alive at some date subsequent to the initial date of exposure, or among those known to be dead. Such an assumption is open to justifiable criticism on the grounds that it may be easier to get information about a man who is alive than one who is dead. Other assumptions suggest themselves ranging from that of early death after rejection in declined risks on the one hand to the assumption that mortality in the unaccounted for group might exceed the observed mortality by only a small percentage, on the other hand. That the mortality would be equal to or less than that of the observed group is impossible to prove.

Obviously the effect of the different assumptions (were they borne out in fact) upon the uncertainty increases with the proportion of unaccounted for cases in the study. If out of 1,000 cases 100 are known to be dead, 895 known to be alive and five are unaccounted for it makes little difference whether those five are assumed all to have died at the outset of the period of observation or to have survived to the end of the period. The total deaths would not be changed by more than 5% and the mortality ratio would not vary by more than 5% of itself. If on the other hand, out of 1,000 cases 100 are known to be dead, 500 known to be alive, and 400 are unaccounted for, the uncertainty as to the mortality in the lost or unaccounted for group becomes of decisive importance.

3. The fact is demonstrated that not only can a study be made of groups of impairments seemingly uninsurable which develops interesting and useful data in respect to correlative conditions and other information by tabulating mortality data based on years of exposure, but also that this kind of study, if pursued in other impairments, might be productive of similar results.

4. Casually, it is indicated that we have been more successful in eliminating the undesirable than we have been in including the possible insurable risk on substandard plans with safety to a company and benefit to an applicant. This suggests the thought that we may, in the future, be able to combine more refinement and bet-

ter technique in our methods with less perfunctory and arbitrary judgment.

5. From the study of the material collected it is apparent that the element of anti-selection is distinctly in evidence.

In this connection, we are reluctantly constrained to conclude that we are not helped as much as we would like to be by our clinical friends. The frequency with which we receive reports as to the harmless character of a perfectly well established glycosuria leads us to wonder whether the knowledge of an assumed harmlessness is mathematically as well as clinically sound, or whether it is the opinion of prognosticians of unquestioned ability based on insufficient data as indicated in section two of these notes. The exposure in the given experience of any one physician or clinic, by virtue of the inconstant relationship between them and groups of patients, must be conspicuously illustrative of missing evidence no matter how complete may be the data in the collected evidence.

To put it another way, it is probable that data collected in, for example, great numbers of cases showing considerable longevity in groups of so-called renal glycosuria would be materially modified by the uncollected evidence in the missing data if it were available, especially as it seems to be the case that death from diabetes is ultimate rather than immediate.

It is worthy of note that, out of 160 deaths among those rejected, 47 would have been claims within one year had they been accepted. Of these 47, there were 35 who were rejected on the evidence of blood sugar time curves, and in an analysis of those dying within a year, evidence is not lacking indicative of a marked selection against the Society, as would be expected.

6. Quite apart from the statistical value of this investigation, we have been impressed with the salutary effect of writing letters to those in the Not Taken Out and Decline Group. It has been rather surprising to find how many people are pleased with the idea that they are not forgotten, and it is equally surprising to find how the subject's interest in himself and his condition increased after a study of a glycosuric condition, and how, by means of frequent observations made by family physicians, a better understanding of their problems follows.

This is quite apart from the salutary effect that among these same people there is an inclination to avail themselves of subsequent studies by the insurance company, which in some instances reveal the maintenance of the same or even a better condition than that assumed when originally observed.

This, of course, applied only to those living; the mortality figures above shown, however, indicate the rather surprisingly high death rate that is found in the group as a whole.

Subsequently there were received 98 additional replies and among these were 18 deaths but this material is not included in Tables VI, VII, VIII and IX. However, it raises the percentage of replies to nearly 94% and the number of deaths tends to strengthen the assumption made regarding the adjusted mortality ratios for the lost cases.

Causes of Death

The study of death claims is rightly criticized as a study from which we derive useful mortality information, but a study of a group in which there are deaths incidentally is a quite different thing. Therefore, we may allude to the causes of death in this group, not necessarily with the idea of having it influence our ideas as to mortality, but in order to coördinate our impressions as to possible correlative conditions.

TABLE IX

Cardio Vascular

Apoplexy	10
Arteriosclerosis	3
Coronary Artery Disease	23
Heart Disease	9
Hypertensive Heart Disease	2
Myocarditis	12
TOTAL	59

Other Causes

Accidents	12
Acute Indigestion (Heart?)	1
Acute Poliomyelitis	1
Appendicitis, Gangrenous	1
Blood Poisoning	2
Bronchopneumonia	1

TABLE IX—Continued

Cancer	8
Cellulitis of Thigh	1
Cirrhosis of Liver	3
Complications (? cause-heart, etc.)	2
Diabetes	28
Diabetic Coma	2
Erysipelas of Scalp	1
Fish bone lodged in intestine	1
Gangrene (Diabetic?)	3
Glioma of Brain	1
Hernia, Strangulated Umbilical	1
Influenza	1
Intestinal Obstruction	1
Jaundice	1
Liver, Abscess Multiple	1
Murdered (stabbed)	2
Natural Death	1
Nephritis	2
Oedema, Pulmonary	2
Peritonitis, General Suppurative	1
Pneumonia	4
Sudden Death	1
Suicide	10
Testicle, Teratoma of Left	1
Tumor of Brain	1
Tuberculosis	1
	99
Unable to ascertain cause of death	16
GRAND TOTAL	174

As might be expected, cardio-vascular disease was of frequent occurrence; and, as noted by many observers, accident and suicide were conspicuous; while diabetes as a basic cause of death remains the greatest single contributor in all the described pathology.

If both clinical and insurance observers could be encouraged to come in contact with each other more frequently, the mortality in glycosuria would be better understood from the standpoint of whether this term is expressive of a so-called harmless anomaly, a prediabetic condition or true diabetes.

The above causes of death are those furnished in the death claim papers for insurance purposes and include certificates required under the regular civic regulations. They must be, therefore, taken for what they are worth. It is arguable that, among the 59 deaths above reported from circulatory conditions, some admitted into

this group were not properly included, as perhaps there were some who had associated circulatory impairments in combination with glycosuria itself. However, in the 1954 records above alluded to, there was none who was known to have had a circulatory disease at entry, unless abnormal blood pressure be considered a circulatory disease *per se,* and in this connection there were 426 where abnormal blood pressure was conspicuous.

It is not the intention here to enlarge upon the subject of the causes of death of this group, or to attempt to determine whether abnormal blood pressure was a cause of or a sequel to the metabolic disturbance indicated by glycosuria; and we therefore express no opinion, except to allude to the fact that glycosurics show a predilection for circulatory deaths.

Comments

If we examine critically the data in table VIII we find that from the figures expressed in Sections 1 and 2 and the percentage in Section 3 that we have an aggregate collection large enough to minimize the influence of the small number of cases which are definitely lost.

Referring to Section 4, we find the exposure years are sufficiently large to allow us to arrive at certain mortality deductions with confidence in view of the comparatively low probable error.

In Section 5 we find that the percentage of those dead to those living is extremely high, which is reflected in the mortality as expressed by Tables VI and VII.

In respect to age distribution we find approximately the same spread as that among insured lives.

Referring to the schedule "Causes of Death", Table IX, we find as would be expected, that cardio-vascular disease plays an important part.

Unfortunately the number of blood sugar time curves obtained is not as high in proportion to the whole group as we would have liked. This happened because a very fair number of cases, especially those where the amount of insurance was small, was selected on the basis of urinalysis alone.

It appears with a high degree of certainty that the formula in use by the Society definitely eliminates the risk which is wholly undesirable, and that, possibly with slight modifications, it is successful in selecting other risks on a proper basis.

DR. BROWN—I ask Dr. Llewellyn Hall, of the Phoenix Mutual Life Insurance Company, to open the discussion.

DR. HALL—The authors are to be congratulated upon the presentation of a mortality study of glycosuria so well substantiating their company's method of selection. It should be a source of genuine satisfaction to Dr. Brathwaite and Dr. Beckwith, whose previous papers attest their long interest in the glycosuria problem, to be able to present, with Mr. Mills' assistance, this satisfactory experience. The paper is a scientific work worthy of the tradition of the Association, and deserves our careful attention.

The homogeneity of the material seems to satisfy the statistical requirements in this regard, with the single important exception, of which the authors are well aware, that some of the cases have had blood sugar studies done and some have not. When sufficient material has accumulated, doubtless a study will be forthcoming, enabling us better to evaluate the role of blood sugar studies in selection.

The size of the standard group, with 234 deaths, warrants placing considerable reliance on the results, which are in accordance with the prevalent belief that the case with accidental glycosuria can be insured safely at standard rates. The Equitable formula has included at standard rates, in addition to the accidental glycosuria cases, certain cases classed as occasional or intermittent.

When we consider the size of the substandard group, the subgroups are unfortunately small and a number of the questions raised by the authors might be explained on the basis of chance fluctuation alone. It is also possible that the original formula is at fault in its severity at the upper limits, as the authors suggest, in so far as it applies to the substandard cases selected.

An important point emphasized by the substandard study is the bad trend of the mortality. This is shown in Table V where the

material is divided according to policy years. This trend is even more serious than would at first appear, because it is contrary to the trend of most substandard business, as reported by Dr. Arthur Hunter in his paper on Heart Murmurs in the Transactions of the Actuarial Society of America for 1936, Volume XXXVII, page 366. In support of the figures developed in connection with this bad mortality trend in glycosuria, the paper on Intermittent Glycosuria, presented by Dr. Bradshaw and Dr. Carber in 1928, is recalled. Their entrants showed "an extremely favorable mortality in the first five policy years," whereas, "policy years six and over are 25%, and policy years eleven and over are 52% in excess of the company's general mortality." An increase in the mortality ratio with duration is also observed in the M. I. S. under symbol 620.

The data on rejected cases give us the benefit of what must have been a stupendous task, requiring infinite patience and perseverance. A group so large as to include 160 deaths, compared with 46.64 expected, warrants placing a considerable degree of dependence on the results. Declinations were "primarily because of glycosuria or hyperglycaemia or both," except that there was a substantial number "where abnormal blood pressure was conspicuous." The case histories of the deaths would in themselves make an interesting study and report. The authors discuss the possible statistical error in such studies arising from the fact that it seems humanly impossible to follow up 100% of cases in such a series. Reference is made in the aforementioned article in the Transactions of the Actuarial Society to this same problem which was faced by Dr. Oscar H. Rogers and Dr. Arthur Hunter in their study of declined heart cases made as long ago as 1892. In that study it was assumed that the mortality of the cases not traced was equal to twice the mortaliy of those traced.

In making practical use of the figures on declined cases, it is perhaps more important to bear in mind an obvious medical consideration, rather than to lay too much emphasis on the mathematical aspects. I refer to the fact that in considering a disease, the outcome of which is so dependent upon treatment as is the case with diabetes, the mortality figures will vary tremendously,

depending upon the per cent. of the particular group studied which has followed proper treatment. Dr. Joslin and his associates, together with Dr. Dublin and Mr. Marks, have made numerous important contributions of a statistical nature toward a better understanding of diabetes. Their figures, which are perhaps most comparable to the mortality of declined cases reported in the paper before us, are those on the mortality of males for years 1920-1929, "discovered early by life insurance examinations." It will be noted that these figures of Dr. Joslin are for a decade that includes a few years of the pre-insulin era. On the other hand, proper treatment in the Equitable series has probably lagged behind the treatment of Dr. Joslin's group. Ages 15-44 showed 26.2 deaths per thousand, ages 45-64 showed 43.6 deaths per thousand. If one may be permitted to compare these death rates with the death rates for ages 35 and 55 according to the American Men's Ultimate Table, the mortality for the two groups would be, very roughly, 548% and 250%.

To demonstrate the room for improvement even among his own patients, Dr. Joslin recently cited the mortality of his physicians with diabetes, which varies from less than one-fourth to less than one-half of the mortality of all his diabetic patients, in age groups under 40, and 40 to 59, respectively. Thanks to protamine insulin, which marks the dawn of a new era in the treatment of diabetes, a study will soon be published, I am told on good authority, proving that a better mortality of his patients is already an actuality.

Nor will improvement be confined to Dr. Joslin's group, for last June he made the estimate that "probably 70,000 more diabetic patients in this country are using insulin today than there were a year ago,"—leaving an estimated number of "one or two hundred thousand persons with mild diabetes in this country who ought to be prevailed upon to take insulin in the early stages of the disease."

Our clinical knowledge of diabetes has led us to expect an increased mortality in some of the glycosurics similar to that now demonstrated as an actuality for the declined group. And the bad trend of the mortality in the substandard group may have

its counterpart in the clinical conviction that the mild diabetic, when neglected, tends to become severe. The underwriting methods of all companies have doubtless been influenced by these clinical conceptions of diabetes. The mortality study now reported by the Equitable has expressed some of these convictions in numerical terms and demands that we review our own underwriting methods in the light of their results. The figures which have been presented have much to offer all of us, including those whose underwriting methods differ markedly from those of the Equitable.

It is the practice of the Phoenix Mutual Life Insurance Company to call for blood sugar determinations, fasting, one-half hour, and two hours after 100 grams of glucose, regardless of the amount of insurance, in all cases in which a specimen two hours after a hearty meal contains as little as 0.2% proven glucose. With a favorable blood sugar curve we generally issue standard insurance, but we rate some cases on account of the glycosuria factor. The small size of the group has not warranted subjecting this practice to the test of a mortality study. If we have erred, it has been probably on the side of conservatism. It may be of some interest to note that by declining cases with a high half-hour reading and cases which fail to return to 120 milligrams at the end of two hours, even though the reading may have returned to normal later, as well as by declining the frank diabetics, the declinations in a series of 150 blood sugar tolerance tests approximate 33%. This compares with the 34% of declinations in Dr. Brathwaite's series of 1,007 blood sugar cases reported in the Proceedings some years ago. He does not include in his declinations 27 per cent., classed as so-called "pre-diabetics," which we would have declined. It would be interesting to know the indications for the blood sugar tests in this old Equitable series, so that we might better estimate the per cent. of abnormal blood sugars that might have been discovered in their substandard group, had blood sugars been done on all entrants.

The general underwriting practice of the Equitable has been described in the Proceedings and should be carefully studied for a complete understanding of their results. When blood sugar

tests have been done, the micro-method of Folin has been used since 1929. The possibility of slightly higher readings when capillary blood instead of venous blood is used should be borne in mind when we interpret the application of their formula to those cases which have had blood sugar time curves done.

By their willingness to underwrite on the basis of urine sugars alone, when small amounts of insurance are involved, the Equitable has undoubtedly made is possible to offer insurance to a larger per cent. of glycosurics than do those of us who require blood sugar studies in a greater proportion of the cases. If the per cent. of blood sugar determinations made in the *T* and *U* groups is typical of the substandard group as a whole, 77% of the substandard cases have been underwritten on the basis of urine sugars alone. A statement from the authors as to whether this per cent. is probably typical of the substandard group as a whole would be helpful in interpreting their results.

Whatever the exact per cent. may prove to be, the company which underwrites substandard cases on the basis of urine sugars alone probably insures an appreciable per cent. of frank diabetics in the group. And it is still a possibility that the substandard group as here reported might, with the aid of blood sugar studies, have been appropriately divided into two groups, one with a mortality but little above normal, and the other with an excessively high mortality. In fact, the "analysis of the causes of death" which the authors have given, enumerating the deaths due to diabetes by policy years, suggests how great a mortality saving might have been made in the total substandard group had the cases dying from diabetes been wholly anticipated and eliminated at the time of original selection. It will be observed that ten of the 97 deaths in the entire substandard group were due to diabetes. Had the diabetic cases been eliminated by blood sugar studies at the time of application the mortality of the substandard group would have been well within the Equitable's standard limits; i. e., considerably less than 130%; and the trend of the mortality would have been more nearly normal.

On the other hand, this small number of deaths due to diabetes, only ten in the space of eleven years, has other important bearings.

It has made possible the satisfactory substandard experience reported. And it lends support to the Equitable's practice of making blood sugar studies obligatory only in "amounts upwards of $10,000 total at risk." As time goes on, it may be that more and more of the insurance public will demand blood sugar studies for their own best interests whenever the suspicion of diabetes has been aroused by the discovery of glycosuria. Obviously, from what has been said, my own feeling would favor making blood sugar studies obligatory for limits lower than $10,000. But the Equitable has an irrefutable answer to any criticism that might be offered to its method of underwriting glycosurics. The method has thus far worked.

The Association is indebted to the authors for their thorough study. I am sure that their researches will be continued toward the goal of a still better understanding of the problem of glycosuria from an insurance point of view.

DR. BROWN—Dr. Francis H. McCrudden of the New England Mutual Life Insurance Company has contributed to our Proceedings in the past relative to this very important subject, and I ask that he continue with this discussion.

DR. MCCRUDDEN—I should like to thank the authors for the courteous invitation to discuss their paper and to express hearty appreciation of their work. Their investigation of declined cases is the only one, so far as I know, in which the sample was large enough and the unknown residue small enough for conclusions to be reliable.

I have no criticism of the work of the Equitable. The differences in the methods of the Equitable and the New England Mutual in underwriting "sugar" cases invite discussion but the differences are so great that a point to point comparison and contrast are impossible. The Equitable selects chiefly on the basis of the urinary findings—glycosuria history, glycosuria findings on examination, findings on urinary sugar tolerance test. The New England Mutual selects on the basis of the findings on a blood-sugar tolerance test. The basis of selection is entirely different in the two cases. In

those cases in which the Equitable does require a blood-sugar tolerance test, the Equitable examines capillary blood; the New England, venous blood. The Equitable requires analysis of several specimens of blood; the New England, but one. The two companies administer different amounts of glucose; they use different analytical techniques. The sugar tolerance tests are too dissimilar for comparison. The Equitable has accepted as standard a little better than one-third of all sugar cases submitted and as substandard about the same proportion. The New England has accepted as standard about four-fifths of all cases submitted. The groups selected by the two companies are not comparable in composition. The two companies have used different standard mortality tables. The mortality figures are not comparable. Significant as these differences are, they are unimportant in comparison with the more fundamental difference in the two cases in point of view as to principles of selection. This fundamental difference, we can compare and contrast.

The Equitable bases selection on the amount of glucose in the urine, the frequency of its occurrence, and the urinary glucose tolerance test findings. Applicants are accepted for standard or for substandard insurance or rejected. A blood-sugar tolerance test is required only in the case of applicants for large amounts.

The New England Mutual bases selection on one postprandial blood-sugar finding. A blood-sugar tolerance test is required in all "glycosuria cases." Those showing not over 0.120 per cent. blood-sugar two hours after ingestion of 100 grams glucose are accepted for standard insurance; the rest are declined.

The Equitable method of classifying risks postulates that excess mortality is a function of *degree* of impairment. Increasing increments of impairment are held to predicate a corresponding increase in mortality. The oftener sugar is found and the more that is found, the greater the risk. Selection is based on *quantitative* differences in impairment.

We, of the New England Mutual, look upon glycosuria as a superficial manifestation of some more fundamental things. We believe that 0.120 per cent. blood-sugar, two hours after the in-

gestion of 100 grams glucose, is a good practical dividing line between types of glycosuria which are different in nature and in underwriting significance. Our method of selection is based on *qualitative* differences in impairment.

So far as passing our test is concerned, we have found that it makes very little difference whether glycosuria has occurred frequently over a long period or only once; whether the finding was recent or remote in time; whether the amount of sugar was large or small. Four reports of glycosuria were no more significant than two reports; 0.6 per cent. glycosuria was no more significant than 0.3 per cent. The underwriting significance of a little sugar in the urine like the underwriting significance of a tiny cancer is not lessened by its littleness. About half of those with definite, *reported* history of diabetes passed the test. Many individuals with a single finding five to ten years previously failed to pass. Half of those who passed showed glycosuria after glucose ingestion. As a basis for selecting risks which we considered standard, these urinary findings after glucose ingestion were no more reliable than the toss of a penny. Quantitative differences in urinary sugar findings, it would appear, do not have the same significance as quantitative differences in hypertension and overweight.

Before discussing the correctness of the statements, (1) "a diagnosis of diabetes cannot depend entirely upon blood-sugar observations," (2) "a normal blood-sugar level three hours after glucose does not rule out diabetes," and the reference to (3) "the fallacy of accepting a single observation two and a half hours after administration of glucose in which some diabetics would be missed," I should want to be convinced of their pertinence. Diagnosis is a clinical problem relating to individuals; selection an underwriting problem relating to groups. Individual life duration is a capricious, indeterminate characteristic. No amount of diagnostic experience with individuals will enable the clinician to predict the life duration of any one individual. In the composite picture of a homogeneous group, differences tend to disappear. The average characteristics of successive samples of a group tend to be alike. The life duration, mortality, of one sample approximates that of any other sample. Experience with one sample enables the under-

writer to predict that of any other sample. The clinical guess as to individual life duration based on diagnosis and the accurate underwriting appraisal of group mortality based on actuarial experience have little in common. Clinical diagnosis and underwriting selection are entirely different problems. It is difficult, however, for clinically trained physicians, even us medical directors with our top dressing of medico-actuarial experience, to get entirely away from the clinical point of view. Hence comes all the irrelevant discussion regarding the diagnosis of renal glycosuria, diabetes innocens, potential diabetes, and true diabetes—all clinical topics and largely matters of definition.

I agree with the authors that in cases having blood-sugar findings the weight given to the urinary findings in their formula is too heavy.

None of the claimants in our group died of diabetes. This does not support the belief that all "glycosuria groups" show an excess of diabetic deaths.

I cannot agree on the desirability of all the companies employing the same technique.

You may be interested in a brief summary of our New England Mutual experience. Between August, 1923, and January, 1936, we made 3,005 tests with the following results:

Accepted Applicants	Amount	Declined
2,288	$24,344,000	616
78.8 per cent.		21.2 per cent.

—Total 2,904—

101 applicants with acceptable tests were declined for other reasons.

Claims (experience carried to July, 1937)

Lives —64

Policies —92

Amount—$1,081,000

This is 116.6 per cent. (amount-basis), 98.2 per cent. (policy count, using amount-basis as standard) of the new England Mutual's own experience. It is equivalent to about 90 per cent. (amount-basis) of the American Men's Select Table. We do not have figures for the mortality by lives. The figures are within normal limits.

Suicides and other violent deaths in our sugar group have been excessive. After making due allowance for the calendar years in which the deaths took place, and the age distribution in the sugar group, we find that the total violent deaths in the sugar group are approximately twice as many by lives as the general business would show for a like exposure. The suicides alone were approximately three times the rate for the general business. The average claim, especially from suicide or other violent deaths, has been exceptionally large. Consequently, even under the pressure of competition, we are not following those companies which issue double indemnity to "sugar cases."

DR. BROWN—In Philadelphia, when one thinks of glycosuria and its scientific aspects, one thinks of Dr. Edward S. Dillon, who is Assistant Professor of Diseases of Metabolism at the University of Pennsylvania Graduate School of Medicine; Chief of the Metabolic Division, Philadelphia General Hospital; and Assistant Physician, Pennsylvania Hospital. He is also associated with the Penn Mutual, for which fact I congratulate that company. Dr. Dillon, will you continue the discussion of this paper?

DR. DILLON—Our Association is indeed fortunate to receive this further communication giving us the benefit of the Equitable Life Assurance Society's experience with applicants showing glycosuria during the four years since Dr. Brathwaite's communication at the meeting in 1933. Dr. Brathwaite has been a pioneer in the study of glycosuria, and his many years of careful research are certain to increase our knowledge of this condition. There is a great deal of material in this paper which not only is of interest to those in life insurance medicine, but which also should command the attention of clinicians as well.

The authors make mention of the fact that opinions are seldom in agreement as to the classification of glycosurics. They suggest that there is a broad twilight zone between the cases having glycosuria, now harmless and always to be harmless, and the cases which are already definitely diabetic, though probably only mildly so. How many of these are destined to become diabetic and to

die ultimately of some diabetic pathology? I am sure it is the experience of most of us that, when evidence of insurability is requested from the physician attending an applicant having "preclinical glycosuria" (as the authors term it), the physician gives us to understand that he regards the glycosuria lightly. Often, the case has been insufficiently studied to be so lightly regarded. Much too often, we receive the diagnosis of "renal glycosuria." How often this diagnosis is warranted depends in large measure on what criteria are used in defining this condition. Joslin, on page 556 of the 1935 edition of his textbook, states that the diagnostic standards require:—that there shall be glycosuria without hyperglycemia; that glucose and not some other copper-reducing substance shall be found in the urine; that every specimen of urine shall contain glucose, whether voided in the fasting condition or after a meal; that there shall be no symptoms of diabetes; and that there shall be no progression toward true diabetes mellitus over an observed period of at least three years. In 13,000 cases, of which 1,659 had non-diabetic glycosuria, he made the diagnosis of renal glycosuria only 44 times.

The authors have commented upon the increase of the mortality with the increasing time of exposure and are of the opinion that this increase will continue. This would seem to be likely from the nature of the case. Probably not more than a very few of the cases were diabetic at the time they were insured. Time is required for the development of the diabetes. Before the discovery of insulin, over 60% of all diabetics died in acidosis. During the insulin era, arteriosclerosis has replaced coma as the chief cause of death. This takes time to develop. It occurs chiefly in diabetics whose diabetes is mild, but has been uncontrolled over a period of years. As Shields Warren has pointed out, the pathology of diabetic arteriosclerosis is different from that of senile arteriosclerosis. It consists of a great thickening of the intima, very much reducing the size of the lumen. It affects muscular arteries, as contrasted with elastic arteries. The arteries particularly affected are the coronaries and the arteries of the lower extremities. It is this pathology which accounts for the large

number of diabetics who die from coronary heart disease and from gangrene of the feet.

That the actual mortality in both the standard and substandard groups should so closely approximate the predicted mortality must be a source of great satisfaction. Why the irregularities of mortality in the substandard groups exist, to which the authors call attention, I have no idea. Probably these will be smoothed out, to some extent at least, with further duration of time. It is of some interest to note that, in the three age groups, the percentage of substandard years of exposure, compared to the total substandard and standard years of exposure in each age group, increases somewhat with the age of the group,—being 29% for the group 39 years of age and under; 32% for the 40-49 group; and 36% for the 50-and-over group.

The authors do not state what proportion of the cases had blood sugar time curves. In discussing the mortality of the *T* & *U* group, they do state that, out of 686 cases, time curves were done in 156. As this is the most heavily rated group—in which, presumably, the largest amount of sugar was found and in which, therefore, the likelihood of requiring time curves was greatest—we are required to assume that the proportion of cases having time curves in the entire series is not greater than for the *T* & *U* group. If this is the case, the number of policies issued on the basis of urinalysis alone is much greater than the number issued on the basis of time curves; and the accuracy of the predicted mortality is all the more remarkable. I, for one, look forward with great anticipation to the time when the number of deaths which will have occurred among the cases having had time curves will be sufficient to enable the authors to compare the mortality of this group with that of the cases selected on the basis of urinalysis only.

The follow-up study of the cases which were declined or not taken out is most interesting. The authors urge us rightly not to become complacent and to be constantly on the alert to discover evidence of insurability. It would seem, however, if we are to go fishing for further evidence of insurability in troubled waters showing a mortality of 467%, that we have our work cut out for us.

I have attempted to make an analysis of the causes of death as given in Table IX and to compare these with the causes of death in patients on the wards of the Metabolic Division of the Philadelphia General Hospital. As the authors point out, cardiovascular deaths predominate. It is rather unfortunate that, in 28 cases, no further data were available than that death was caused by diabetes. Diabetics do not die from glycosuria, hyperglycemia, or faulty carbohydrate metabolism (that is, diabetes). They die only from complications which follow the abnormal carbohydrate metabolism. The complications may be intimately dependent upon the abnormal carbohydrate metabolism, as, for instance, diabetic coma, which comes about because of faulty fat metabolism, which is directly dependent upon faulty carbohydrate metabolism. The connection between diabetes and arteriosclerosis is intimate, but less so than is the case with coma. Pyogenic infections and tuberculosis are less directly connected. And so the list goes until we come to causes of death which have no connection with diabetes whatsoever. It is always desirable to determine precisely the exact mode of exit.

During the six years 1931-1936, there were 3,009 patients admitted to the wards, 521 of whom died while there. A comparison between causes of death in our hospital cases and in Table IX is as follows:

		P.G.H.	Table IX	
Arteriosclerotic	Heart	106	51	
	Gangrene	131	3	
	Apoplexy	20	10	
	Nephritis	18	2	
	Acidosis	59	2	
	Infections	104	16	
	Cancer	29	11	
	Tuberculosis	14	1	Accidents 12
	Miscellaneous	40	34	Suicide 10
	Diabetes	.....	28	
		521	158	

There is much that I might comment upon in comparing these groups. The large number of gangrene cases in the hospital group is striking. Gangrene is much more prevalent in the under-

privileged classes, whose diabetes had been long uncared for; whose feet are not kept clean; whose corns, calluses, ingrowing toe nails, and various traumata, are neglected. To a less degree, the presence of a large number of cases of acidosis in the hospital group is to be noted, and this, too, is significant of ignorance and neglect. Perhaps, in the insurance group these two causes of death are obscured to some extent in the cases recorded as having died of simply "diabetes." The number of cases in the hospital group which died of infections is also high. Fatal infections in diabetics occur chiefly in three locations,—the respiratory tract, the urinary tract, and the skin. On the other hand, the deaths in the insurance group are relatively numerous in the heart group and in the miscellaneous group, where 22 deaths occurred from accidents and suicide. My experience with private patients compares much more closely with that of the insurance group than of the hospital group. There have been no deaths from coma or gangrene. More than half of the deaths in my private cases have been heart deaths. Coronary occlusion has been a very common cause.

I should like to congratulate the authors for this very valuable and stimulating follow-up investigation.

DR. BROWN—Dr. Beckwith, will you close the discussion of this paper?

DR. BECKWITH—It is quite true that the higher percentage of blood sugar tests increases the homogeneity of the group. Without extracting the figures accurately it is our belief that we have obtained glucose blood sugar tests in between 20 and 25% of the insured business.

In view of the development of protamine insulin now coming into common use, the mortality should doubtless be improved.

It would appear that the practice of the Phoenix Mutual is conservative, inasmuch as the so-called "pre-diabetic" cases mentioned, as referred to by Dr. Brathwaite in 1928, were actually cases issued by the Equitable at ratings upward of 150%. The indication in the Equitable for carrying out the blood sugar test

has always been that if a sugar record existed or glucose was found on examination the test was made if more than $10,000 at risk (total). We have no means of estimating the percentage of abnormal blood sugars that might have appeared if all the insured lives had been studied with blood sugar tests. It is quite probable that blood sugar studies might have improved some offers in the substandard groups. Conversely others might have been declined.

The adverse effect of the possible inclusion of frank diabetics in the substandard group where blood sugar tests were not done is perhaps offset to some extent by the inclusion of some low-threshold cases who could have demonstrated their fitness for better offers if a test had been done.

Dr. McCrudden's remark is rather surprising,—that the New England Mutual is able to issue standard policies to four-fifths of their applicants with glycosuria, particularly when it would appear that they employ a rather severe criterion for the issuance of standard insurance, viz: venous blood showing 120 or below, at the end of two hours, after 100 grams of glucose. In connection with that point, it has always been our opinion that the study of a curve was of greater importance than the valuation of the finding at a single point, our belief in this being based on the fact that the personal equation is bound to enter into the laboratory technique to some extent, and that it was far more important to see where a curve fell with respect to certain defined areas than to attempt to fix any one point at a given time.

With reference to Dr. McCrudden's remarks concerning the quantity of urinary sugar, it would seem that the clinicians are interested because we still hear them talking about having an individual "sugar-free." From an insurance standpoint, we can only say that in the Equitable's mortality study, the larger amounts of urinary sugar appear to have been important because the cases that have been excluded on these grounds alone have given a high mortality as demonstrated by our study of declined and not taken cases.

With reference to his remarks about there being no diabetic deaths in the group, that does not necessarily have any connection with the method of selection because, even in our much larger

group, we had comparatively few so-called "diabetic" deaths, and, therefore, as Dr. Dillon has also pointed out, it is a question whether anyone dies of diabetes at all or whether they die of some other cause related to diabetes.

The question of suicides and accidental deaths is also similar to that encountered in our follow-up study and can possibly be explained on the ground of arteriosclerosis with mental changes. I think Dr. Joslin has also referred to the possibility of accidents being due to beginning acidosis.

The figures presented by Dr. Dillon from the Philadelphia General Hospital are interesting, especially so since they show a higher percentage of cardio-vascular deaths than our series, presumably associated with some metabolic disturbance, although it is not stated that this was diabetes. The obvious difficulty in comparisons of this sort is that the diagnosis in an institution is often more correct as to the cause of death, whereas we were compelled, not infrequently, to rely on the cause of death stated on the death certificate or obtained from lay sources.

In closing, we again wish to emphasize:

1st That in spite of much opinion to the contrary, the double criterion of blood and urine findings is important, and that blood findings alone should not be relied upon.

2nd That glycosuria is a relatively common impairment and yet is one in life insurance medicine that presents great diversity of opinion, both as to obtaining the data and underwriting the risk on given data.

3rd In view of this, it would seem extremely desirable for all companies to obtain some method which can be standardized—at least to a far greater extent than the present method of underwriting these risks.

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Dr. Brown—I should like to interrupt the scheduled procedure of the meeting for a very few minutes.

Two years ago, there was held in London an International Congress of Life Insurance Medicine. The outstanding exhibit and the outstanding paper at that Congress had to do with health

conservation. It was my privilege at that time to meet a very kind and lovable soul who was responsible for that part of the program. He is an individual who, in Europe, has done more toward the advancement of periodic examinations and health conservation than any other person. He had charge of all of this work in Germany.

He has come to America and his great interest in life insurance and its kindred problems has caused him to come here today. I would like to introduce to you Dr. Otto Neustatter. I wish to welcome him to America and to this meeting, and I would like to have him come to the platform and say something to us.

DR. NEUSTATTER—Mr. President and Gentlemen: I feel very much moved by the kind words that your President has just said about me. I have been blushing to match the red color in your banner. It is a great privilege and a great pleasure and it is of great interest to me to be here and attend one of your meetings, which are not unknown to me. I have studied the protocols of these unique meetings and I can say that I have taken from them a great deal of information.

I admire the scientific spirit that prevails in your work, and I can say that we in Germany, who otherwise are also known to be ahead in scientific matters, have not been able, so far, to put before the profession anything on life insurance medicine in the manner in which you have.

The work that you have done here, as I say, is not unknown to me, but the kindness with which I have been received and with which my work has been mentioned is overwhelming. I wish to thank your President for the kind words he has said and I only hope to be able to take more away from you than I have brought to you. I am sure that the furtherance of life insurance medicine is a common interest that we all have.

DR. BROWN—It is very pleasant to see you again, Dr. Neustatter. Thank you very much!

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DR. BROWN—Both authors of the next paper have very kindly and with much profit to us contributed to our Proceedings on a number of previous occasions. Many of us know them personally, and possibly a formal word of introduction is unnecessary.

Those of you who may visit the World's Fair in this City in 1939 will find there much for which Dr. Louis I. Dublin will have been responsible, as he is Chairman of the Committee of Medicine and Public Health for that exhibition. He has been President of the American Public Health Association. He is Third Vice-President and Statistician of the Metropolitan Life Insurance Company.

Mr. Herbert H. Marks, the other author of this paper, is Medical Statistician of the same company. He was with the Bureau of the Census in 1919 and 1920 and since then has been a joint author of at least ten papers on medical subjects treated from a statistical viewpoint.

Dr. Dublin, will you speak to us on the subject "The Mortality of Women According to Build."

THE BUILD OF WOMEN AND ITS RELATION TO THEIR MORTALITY

A PRELIMINARY REPORT

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The insurance business has moved a long way from its original attitude of fear and doubt toward women as policyholders. In most companies during the past two decades, the volume of life insurance on females, both in number of policies and amount, has, in fact, increased at a much faster rate than insurance on men. Women now form a large and increasing proportion of our insurance clientele. From almost every point of view, they are today desirable insurance risks, and with reasonable care in their selection, the companies accepting them need have no fear that they will have an unfavorable effect on the company's general mortality experience. It goes almost without saying, however, that women as applicants for life insurance present specific problems of underwriting. The practise of the life insurance business in this regard has been a rule of thumb extension of the procedures applicable to men. This has served well enough, but with the rapidly accumulating experience on women, it is time that this experience were analyzed with a view to a more informed treatment of female applicants.

One very important factor in medical underwriting, one which took the insurance business a long time to learn, is the relation of build to mortality. In evaluating build among females so little authentic data have been available that the tables used are the tables on male lives or a simple modification of them. These tables were based on the Medico-Actuarial Mortality Investigation and the 1918 report on build of the Joint Committee on Mortality of this Association and the Actuarial Society. In the former investigation, the data on women were tabulated and

analyzed, and while certain tendencies emerged from this study, in the words of the report, "the statistics on the influence of build on mortality of women enabled the Committee to deduce little of substantial value." The meager results of this 1912 study served to discourage the Joint Committee eight years ago when the Medical Impairment Study was being made. Consequently, the build study, made at that time, was limited to male lives. It was in order to fill this important gap in our knowledge that the present study was undertaken. As part of this study it was thought worthwhile to make height and weight tables to see whether the old standards, based on the Medico-Actuarial tables, hold today. There has been much discussion of changes in the stature of the new generation. We have gone through an era in which a craze for reducing has raged among women. We wanted to see if this and other factors had any discernible and lasting effect on the average weights.

Our data are extensive even though they are derived from the experience of a single company. This preliminary report is concerned with our Standard cases, those in our Regular Ordinary Branch (including monthly premium cases). We have used all available records, namely, those for which we had routinely prepared record cards from 1922 to 1934 inclusive. During the first two years 1922 to 1923, a record was made of every policy placed. In 1924 only selected types of risks in the Regular Ordinary Branch were recorded. The classes selected for study contained very few women. From 1925 to 1931 inclusive, record cards were made routinely for one-fifth of all our Standard cases, being an unselected and representative sample. Beginning with 1932, we have recorded approximately 100,000 policies (male and female combined) each year including all issues of $5,000 or more, and excluding monthly premium cases. By this system, approximately one-eighth of all the issues on female lives were recorded. In making up our descriptive tables, allowance has been made for the varying samples used in each year of issue. We checked back to the original application every case in which the height or weight was unusually high or low.

Our descriptive tables are based upon 329,627 policies and our mortality tables on 308,228 policies, the latter figure excluding those cancelled in the year of issue and extremely short and extremely tall women.

Before presenting our findings, a few observations regarding the material are in order. We are dealing very largely with persons whose height and weight were not actually measured at the time of the examination. The facts on height and weight are usually those stated by the applicant. This is especially true of height. The actual weights were more frequently taken in the later years of the experience than in the earlier years. As a consequence, we have the same problem here that has come up in all previous insurance studies of build, namely, a concentration of records in the average height group, generally on all the even inches, and also a high percentage of weights reported in figures ending in zero or five, and secondarily in even numbers. The defect in the record of heights was made somewhat worse by our rule from 1925 on that heights ending in a half inch be recorded to the nearest even inch, i.e., 5′3½″ and 5′4½″ were taken as 5′4″. There may also be a systematic overstatement of weight by those markedly underweight and understatement by those markedly overweight. The measurements of the applicant are presumed to be taken in ordinary street dress, without heavy outer clothing.

Certain other aspects of the material must be kept in mind in judging our results: (1) The type of risk represented by these women insured in the Metropolitan and (2) the internal changes in this group in the years covered by this study. These women are largely drawn from the salaried and wage-earning classes in the urban areas of this country and Canada. They are the wives and daughters in families which are regularly contacted by our agency force. They are, for the most part, housewives, factory workers, stenographers and clerks. While they are drawn from every part of the two countries, there is probably an undue proportion from the eastern seaboard. Again, due to the more intelligent underwriting of female risks in recent years, as well as the increasing proportion which immigrants and their families form in the insurable population, the percentage of women from these

groups among our policyholders has increased in recent years. In like manner, the build ratings for women will have some effect on the results, as for example, the restrictions on underweight at the younger ages and on overweight at the older ages. Similar in its effect is the more careful medical selection of those whose weights border on the Standard limits. Unfortunately, we have no numerical measures of some of these factors, but we should not lose sight of them and the effect they may have exercised on our results.

Height

The mean heights by age in the Standard material are shown in Table 1. The averages rise from approximately 5′3¼″ at age 15, to 5′4¼″ in middle adult life. The maximum height is recorded in the age group 45 to 49. The averages show a slight but persistent fall past 50 years of age. At the younger ages, the increases are fairly large and significant because they represent increments due to normal growth. The differences at the various ages between 25 and 50 are small and are probably of little significance. The averages are approximately the same as those found in the Medico-Actuarial Mortality Investigation, but slightly less than in the Medical Impairment Study.

TABLE 1

Mean Heights of Women by Age

Metropolitan Life Insurance Company, Standard Issues, 1922-1934

Age	Height in inches	Number of cases
15	63.22	7,195
16	63.63	9,255
17	63.85	11,767
18–19	64.02	33,802
20–24	64.07	90,209
25–29	64.09	60,374
30–34	64.17	42,646
35–39	64.24	31,734
40–44	64.22	20,648
45–49	64.26	12,186
50–54	64.12	6,240
55 and over	64.08	3,571

So far as this Standard material is concerned, the average height of adult American women has not changed. We do not find maximal figures at the younger ages which would be indicative of a steady and progressive rise in the average height from one generation to the next. Moreover, a comparison of the average heights by age of the 1922-1923 and the 1932-1934 Standard issues shows no significant increase in the averages except at age 15. Indeed, at ages past 25, averages for the earlier issues tended to be somewhat higher than for the later ones. These results seem to be little modified by our Substandard material. In this respect, our present data are in conformity with the findings of the Medical Impairment Study. Possibly these fundamental data are so defective that we must discard them as a source of information on this question of changing average heights. We think however, they are not as bad as that. We think rather that in the aggregate they are dependable and lead to the conclusion stated above. This may well be due to the fact that our data included increasing proportions of women of foreign race stocks, many of whom were of smaller stature than our native stocks. Moreover, since the averages remain fairly constant, the figures may also indicate that the newer generation of foreign race stocks is tending to conform in stature to the standards of the native stock.

The lowest height recorded among the women in this study was 4′0″; the greatest height, 6′6″.

Weight Characteristics

In Table 2 are given the ungraduated mean and median weights and the standard deviations of these weights around the mean. Throughout most of the table, the data form fairly smooth series. There is a definite and consistent increase in the average weight by age at each inch of height up to a maximum in the early fifties, but beyond this point the averages do not consistently increase.

Although the increases in weight *with age* at each inch of height are consistent, there are notable variations in the size of the increments. Between ages 15 and 16, the increments tend to be larger at the short and at the tall heights than at the middle heights, namely, three to five pounds at the extremes, compared

TABLE 2

Mean and Median Weights of Women and Standard Deviations of the Weights Around the Means. By Age Groups and Inch of Height. Standard Issues Only. Metropolitan Life Insurance Company, Ordinary Department, 1922-1934.

Height	WEIGHT IN POUNDS AT SPECIFIED AGES											
	15	16	17	18-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55 & Over
4′ 8″												
Mean	91.2	*	*	101.6	106.7	107.0	114.0	114.6	*	*	*	*
Median	92.3	*	*	101.2	104.7	105.6	110.0	111.5	*	*	*	*
St. Deviation	11.0	*	*	6.9	12.1	12.3	19.0	15.8	*	*	*	*
4′ 9″												
Mean	94.8	99.3	103.0	104.4	109.9	113.2	113.8	125.8	127.5	*	*	*
Median	93.0	97.1	100.5	102.7	110.0	111.6	114.5	126.8	133.5	*	*	*
St. Deviation	11.2	9.8	9.0	10.2	13.1	14.8	15.1	18.0	17.6	*	*	*
4′ 10″												
Mean	93.3	103.2	101.8	103.2	107.2	112.4	118.5	120.9	126.9	126.7	130.4	127.2
Median	92.2	102.2	99.4	101.3	105.1	110.6	117.9	121.0	129.5	130.1	130.7	127.1
St. Deviation	11.5	13.8	9.7	10.4	13.1	14.8	15.5	17.5	16.1	16.3	14.3	17.3
4′ 11″												
Mean	97.3	101.2	106.3	106.2	107.7	114.6	118.8	125.9	125.6	131.5	126.7	123.7
Median	96.3	102.6	103.8	104.6	104.8	112.1	117.9	125.8	125.6	132.7	126.1	119.4
St. Deviation	9.9	8.7	10.7	10.5	13.6	15.1	15.9	16.4	16.9	15.6	15.8	20.5
5′ 0″												
Mean	102.6	106.3	107.7	110.2	111.6	116.6	121.7	126.4	131.0	132.7	134.5	135.8
Median	101.4	105.0	106.7	108.4	109.1	113.7	120.1	125.8	132.1	134.3	136.0	138.3
St. Deviation	11.5	11.4	10.9	11.9	13.4	15.6	16.3	17.1	16.6	16.4	16.4	17.0
5′ 1″												
Mean	104.9	108.9	109.8	111.8	113.7	119.4	124.6	128.6	131.1	133.1	137.4	135.3
Median	103.5	107.6	109.1	110.3	111.4	117.3	122.7	127.7	131.0	134.2	139.9	135.8
St. Deviation	11.7	11.7	11.1	11.8	13.0	16.0	16.5	17.5	16.9	18.3	17.0	18.1
5′ 2″												
Mean	108.7	111.3	113.5	115.1	116.9	121.5	126.7	131.4	134.5	136.7	138.2	139.0
Median	107.7	110.2	112.5	113.2	114.9	119.2	125.3	131.0	135.1	138.2	139.0	140.3
St. Deviation	11.1	11.4	11.6	12.3	13.3	15.7	16.4	17.0	17.0	17.0	16.8	17.6
5′ 3″												
Mean	112.8	115.6	116.7	118.5	120.4	125.4	130.2	134.6	137.7	140.4	141.5	142.3
Median	111.5	114.3	115.6	116.9	118.5	123.0	128.6	134.0	137.6	141.3	143.6	144.0
St. Deviation	12.3	12.0	11.7	12.5	13.7	16.1	17.0	17.7	17.3	17.6	18.1	18.6
5′ 4″												
Mean	116.3	118.5	120.2	121.8	123.5	128.6	133.4	137.7	141.0	142.5	144.3	144.7
Median	114.6	117.2	118.9	120.3	121.4	126.3	131.4	136.8	140.9	142.8	145.6	145.2
St. Deviation	13.1	13.1	12.8	13.0	14.1	16.3	17.2	17.5	17.5	17.1	17.8	17.1
5′ 5″												
Mean	120.8	121.7	123.6	125.1	127.0	132.3	137.1	141.8	145.3	146.7	150.0	150.9
Median	119.0	120.3	122.2	123.4	124.8	129.9	135.3	141.1	145.0	146.3	150.5	151.7
St. Deviation	14.4	13.2	12.8	13.6	14.8	16.8	17.7	18.0	17.9	17.5	17.8	17.7
5′ 6″												
Mean	123.3	123.9	126.9	128.1	130.6	135.5	141.4	145.7	149.4	151.1	152.6	152.9
Median	121.5	122.4	125.5	126.5	128.4	132.5	139.7	145.0	149.5	151.8	153.9	153.9
St. Deviation	13.9	12.9	14.0	13.4	15.4	17.3	17.9	18.4	18.1	18.6	19.0	17.6
5′ 7″												
Mean	125.4	130.3	131.5	132.2	134.6	140.7	145.2	149.4	152.7	155.3	156.6	158.4
Median	123.1	128.7	129.5	130.4	131.9	138.1	143.4	148.0	152.2	155.8	156.8	157.6
St. Deviation	13.6	14.0	14.9	14.5	16.1	18.1	18.3	19.4	18.6	18.0	18.1	18.0
5′ 8″												
Mean	131.1	134.4	133.8	136.2	139.3	143.2	149.8	154.2	158.0	161.4	164.0	161.5
Median	129.7	132.9	132.1	134.3	136.9	140.3	148.1	154.0	159.4	162.6	167.7	166.3
St. Deviation	14.4	15.6	15.4	15.2	17.3	18.4	19.1	19.7	19.7	18.7	19.4	18.9
5′ 9″												
Mean	131.1	135.7	139.5	139.7	142.1	147.1	156.0	160.5	161.6	166.8	164.3	166.0
Median	129.1	134.0	136.3	136.9	139.0	145.0	154.3	159.8	160.5	169.7	165.1	164.4
St. Deviation	12.6	14.4	16.0	15.1	17.1	18.7	20.1	20.3	19.9	20.1	19.3	17.4
5′ 10″												
Mean	*	143.7	141.8	143.2	147.1	150.1	161.3	164.1	163.4	169.2	171.8	170.8
Median	*	140.3	141.0	139.3	143.0	148.6	160.5	165.4	163.2	172.3	176.9	169.9
St. Deviation	*	17.0	13.6	17.1	18.0	18.8	19.6	20.1	21.0	19.5	19.6	13.5
5′ 11″												
Mean	*	*	*	147.9	150.8	159.0	160.4	165.5	176.8	175.9	*	*
Median	*	*	*	144.7	149.4	159.2	161.0	169.2	177.1	171.6	*	*
St. Deviation	*	*	*	15.9	19.1	20.7	20.6	21.9	18.6	18.4	*	*
6′ 0″												
Mean	*	*	*	*	157.8	163.2	173.1	167.4	*	*	*	*
Median	*	*	*	*	154.5	160.3	178.1	160.8	*	*	*	*
St. Deviation	*	*	*	*	19.4	20.0	21.5	22.6	*	*	*	*

*Less than 20 cases.

to one to three pounds at the middle heights. Between ages 16 and 17, the increments of weight with age are usually one to two pounds and vary less than between ages 15 and 16. Between 17 and age group 18-19, the increments of weight with age are fairly constant at each height, one to two pounds in all but a few cases. This is likewise true between ages 18-19 and 20-24, these increments being two to three pounds in all but a few instances. Between ages 20-24 and 25-29, the increase in the average weight at each inch of height is usually five to six pounds. Approximately the same gain in weight is recorded in the next five years of life at each inch of height. The increments then begin to diminish in size, approximately one pound for each five years of age. Between ages 30-34 and 35-39, the increment is usually four to five pounds; in the next five year interval, three to four pounds; and in the next two periods, two to three pounds. Between ages 50-54 and 55-59, the change in the middle heights, where the data are most abundant, varies from —1 to +1 pound.

There is likewise a consistent increase in the average weight *with increasing height* at each age. These increments are, for the most part, more regular than those with advancing age. Up to 5′3″ or 5′4″, there is usually a three pound increase with each inch of height, and from 5′4″ on an increase of approximately four pounds.

The median weights are generally a pound or two less than the mean weights up to age 35. Between 35 and 45, the two types of averages are approximately the same, but after age 45 the median weights are often higher than the mean weights. The diminishing difference between median and mean, past age 35, is purely artificial and is due merely to the fact that at these ages the extreme overweights are all rated risks. By the very nature of the weight curve the median must be lower than the mean, and when the data for the combined Standard and Substandard are available, this, we believe, will be found to be the case.

Comparison with the Standard (Medico-Actuarial) Table

At ages under 20, our averages generally conform, within a pound or two, to the new averages that were compiled in connec-

tion with the Medical Impairment Study. At age 15, our averages at the very short heights are somewhat heavier, probably indicating that the Medical Impairment Study table at these points was too steeply graduated. In like manner, our averages at the taller heights tend to be somewhat lower than the Medical Impairment Study table, and again it may be due to the artificial nature of the graduation of those results. At age 16 our averages are likewise somewhat higher at the very short heights, but in fair agreement at all the other heights. At age 17, our table is usually one pound heavier than the Medical Impairment Study, but at 18-19, one pound lighter.

At ages 20-24, our averages are consistently lower than those of the Medico-Actuarial table. At ages 25-29, our averages are within a pound or two of the Medico-Actuarial table, up to and including heights 5'10". Beginning with age group 30-34, our averages are systematically higher than the Medico-Actuarial table. At the middle heights, the differences are 1-2 pounds at ages 30-34, 2-3 pounds at ages 35-39 and 40-44, and 1-2 pounds at 45-49 and 50-54. Even larger differences are recorded at the taller heights. At 55 and over, our averages are generally a pound or two below the Medico-Actuarial figures. The comparative figures of the two tables are given in Table 3.

These comparisons are based on Standard data only. In the Substandard are included, at the younger ages, large numbers of rateable underweights and a few overweights. But at the middle and later adult ages, there are comparatively few rateable underweights, but many rateable overweights. Consequently, it is likely that we shall, when all the figures are in, show consistently lower average weights at ages 20 to 24 and appreciably higher weights consistently after age 25. The standard height-weight table is based only on those insured at regular rates of premium and, consequently, tends definitely to overstate the averages at the younger ages and understate them at the older ages.

From this comparison, it is clear that the increases in weight by age during adult life are much less regular than in the standard height-weight table. The actual gains between 20 and 40 are larger than the increments of the standard table, and are smaller past 40.

TABLE 3

Average Weights of Women by Height and Age

Metropolitan Life Insurance Company, Standard Issues, 1922-1934 (Ungraduated)* Compared with Medical Impairment Study (Graduated) at Ages Under 20 and Medico-Actuarial Study (Graduated) at Ages 20 and Over.

	15		16		17		18–19		20–24		25–29	
Height	M.L.I. Co.	M.I.S.	M.L.I. Co.	M.I.S.	M.L.I. Co.	M.I.S.	M.L.I. Co.	M.I.S.	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.
4′ 8″	91	87	....	91	....	96	102	102	107	107	107	110
9″	95	91	99	95	103	99	104	104	110	109	113	112
10″	93	95	103	99	102	102	103	106	107	111	112	114
11″	97	99	101	102	106	105	106	108	108	113	115	116
5′ 0″	103	103	106	105	108	107	110	110	112	115	117	118
1″	105	106	109	108	110	110	112	113	114	117	119	120
2″	109	109	111	111	114	113	115	116	117	120	122	122
3″	113	113	116	114	117	116	119	119	120	123	125	125
4″	116	117	119	118	120	119	122	122	124	126	129	129
5″	121	121	122	122	124	123	125	125	127	129	132	132
6″	123	125	124	126	127	127	128	129	131	133	136	136
7″	125	129	130	130	132	131	132	133	135	137	141	140
8″	131	133	134	134	134	135	136	137	139	141	143	144
9″	131	137	136	138	140	139	140	141	142	145	147	148
10″	....	141	144	142	142	143	143	145	147	149	150	152
11″	....	145	....	146	....	147	148	149	151	153	159	155
6′ 0″	....	149	....	150	....	151	....	153	158	157	163	159

	30–34		35–39		40–44		45–49		50–54		55 and over	
Height	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.	M.L.I. Co.	M.A.
4′ 8″	114	113	115	116	....	120	....	123	....	125	....	126
9″	114	115	126	118	128	122	....	125	....	127	....	128
10″	119	117	121	120	127	124	127	127	130	129	127	130
11″	119	119	126	122	126	126	132	129	127	131	124	132
5′ 0″	122	121	126	124	131	128	133	131	135	133	136	134
1″	125	123	129	126	131	130	133	133	137	135	135	137
2″	127	125	131	129	135	133	137	136	138	138	139	140
3″	130	128	135	132	138	136	140	139	142	141	142	143
4″	133	132	138	136	141	139	143	142	144	144	145	146
5″	137	136	142	140	145	143	147	146	150	148	151	150
6″	141	140	146	144	149	147	151	151	153	152	153	153
7″	145	144	149	148	153	151	155	155	157	157	158	158
8″	150	148	154	152	158	155	161	159	164	162	162	163
9″	156	152	161	156	162	159	167	163	164	166	166	167
10″	161	155	164	159	163	162	169	166	172	170	171	173
11″	160	158	166	162	177	166	176	170	....	174	....	177
6′ 0″	173	162	167	165	....	169	....	173	....	177	....	182

*Averages not computed where number of cases was less than 20.

There is no gain past 50-54 and the increase shown by the standard table is, in our judgment, questionable. Indeed, inspection of the original Medico-Actuarial data confirms this observation.

Comparative Mortality by Build Groups

For the mortality study, we have divided our material into three broad height groups and nine broad weight groups. The height groups are:

Short	4′8″ to 5′1″
Medium	5′2″ to 5′6″
Tall	5′7″ to 6′0″

We have excluded from this part of the study very short and tall women, i.e., those under 4′8″ and over 6′0″.

The weight groups, which represent percentage departures from the average weight, for height and age, of the Medico-Actuarial table, are given below. Here there was a choice either of pound or percentage groups. We judged the latter to be the more suitable.

Underweight
25% or more
15–24%
5–14%
Average Weight
Overweight
5–14%
15–24%
25–34%
35–44%
45% or more

Since we are dealing with Standard issues only, the ratings for build have a considerable effect on the relative volume of the experience at the several age groups at issue. Thus, at the younger ages, the ratings for underweight are more severe than at the older ages, but the reverse is true for overweights. Consequently, the extreme underweight group in the Standard experience was made up largely of older women, while the extreme overweight group was made up almost solely of younger women. For this and other reasons, the mortality ratios at all ages at issue com-

TABLE 4

Mortality of Women According to Build

Ratios of Actual to Expected Deaths by Metropolitan Female Mortality Table, 1922-1936 (Ungraduated). By Age Groups at Issue and by Duration of Insurance. Standard Issues Only.

Metropolitan Life Insurance Company, Ordinary Department, Issues of 1922-1934 Carried to Anniversary in 1936.

Build Group	AGGREGATE										
	Exposed	Per Cent. Actual of Expected Deaths					Deaths				
		All Ages	Under 30	30-39	40-49	50 & Over	All Ages	Under 30	30-39	40-49	50 & Over
GRAND TOTAL	2,015,222	100.0	100.0	100.0	100.0	100.0	6,621	2,929	1,661	1,231	800
Underweight											
25% or more	10,072	90.3	96.6	106.6	90.2	74.5	48	6	14	16	12
15-24%	183,114	96.1	105.0	92.6	86.3	92.3	611	261	154	115	81
5-14%	566,730	98.5	102.0	105.5	81.1	89.2	1,685	930	410	209	136
Average weight	559,857	100.3	99.0	95.4	114.4	94.6	1,776	851	383	344	198
Overweight											
5-14%	392,453	101.1	96.9	97.1	102.6	115.7	1,402	503	345	306	248
15-24%	207,744	100.0	97.0	98.1	107.0	98.1	771	237	220	207	107
25-34%	75,678	117.3	108.0	123.7	116.9	149.0	280	109	123	33	15
35-44%	17,347	93.7	83.2	105.5	*	*	44	28	12	1	3
45% and Over	2,227	*	*	*	*	*	4	4	--	--	--
SHORT HEIGHT											
Underweight											
25% or more	1,493	124.1	*	*	*	*	9	2	1	2	4
15-24%	23,083	120.0	125.9	141.3	104.9	89.8	95	41	27	16	11
5-14%	65,804	109.5	106.2	100.5	122.9	130.6	215	114	44	31	26
Average weight	62,648	99.7	97.6	82.1	134.8	94.3	190	100	30	38	22
Overweight											
5-14%	44,208	102.0	90.5	98.7	130.1	92.9	161	52	39	47	23
15-24%	25,589	123.9	120.6	130.9	102.0	148.3	120	35	39	23	23
25-34%	9,395	129.2	129.2	133.3	118.2	*	39	16	17	5	1
35-44%	2,593	69.1	*	*	*	*	5	4	1	--	--
45% and Over	392	*	*	*	*	*	1	1	--	--	--
MEDIUM HEIGHT											
Underweight											
25% or more	6,850	88.2	*	124.2	99.3	59.7	33	2	11	13	7
15-24%	133,156	92.4	106.1	82.0	80.7	91.1	428	192	98	80	58
5-14%	422,546	97.1	99.1	110.0	77.1	85.9	1,230	679	310	147	94
Average weight	422,328	100.3	96.7	99.9	113.1	97.3	1,336	626	306	253	151
Overweight											
5-14%	291,704	100.3	96.8	98.4	96.5	117.8	1,020	381	254	204	181
15-24%	152,789	97.6	95.4	94.9	107.7	90.0	550	176	149	152	73
25-34%	55,322	115.7	104.5	122.7	123.5	132.9	202	76	90	25	11
35-44%	12,744	99.0	88.2	110.8	*	*	34	22	9	1	2
45% and Over	1,565	*	*	*	*	*	3	3	--	--	--
TALL HEIGHT											
Underweight											
25% or more	1,729	70.3	*	*	*	*	6	2	2	1	1
15-24%	26,875	94.0	79.7	104.7	100.6	101.2	88	28	29	19	12
5-14%	78,380	96.8	114.4	88.8	73.8	69.3	240	137	56	31	16
Average weight	74,881	100.7	113.9	80.1	108.6	80.8	250	125	47	53	25
Overweight											
5-14%	56,541	104.0	102.5	90.1	108.6	122.4	221	70	52	55	44
15-24%	29,366	91.4	84.5	85.2	107.5	88.5	101	26	32	32	11
25-34%	10,961	115.1	107.5	120.0	*	*	39	17	16	3	3
35-44%	2,010	93.6	*	*	*	*	5	2	2	--	1
45% and Over	270	*	*	*	*	*	--	--	--	--	--

*Less than 5 deaths.

TABLE 4—Continued

Mortality of Women According to Build

Ratios of Actual to Expected Deaths by Metropolitan Female Mortality Table, 1922-1936 (Ungraduated). By Age Groups at Issue and by Duration of Insurance. Standard Issues Only.

Metropolitan Life Insurance Company, Ordinary Department, Issues of 1922-1934 Carried to Anniversary in 1936.

Build Group	FIRST FIVE POLICY YEARS									
	Per Cent. Actual of Expected Deaths					Deaths				
	All Ages	Under 30	30-39	40-49	50 & Over	All Ages	Under 30	30-39	40-49	50 & Over
GRAND TOTAL	100.0	100.0	100.0	100.0	100.0	3,307	1,573	813	551	370
Underweight										
25% or more	97.0	*	132.5	76.9	69.6	23	4	8	6	5
15-24%	100.3	109.2	89.7	92.9	102.4	317	147	73	55	42
5-14%	100.5	100.2	110.3	88.7	94.8	891	505	214	103	69
Average weight	97.9	99.4	91.3	98.5	103.3	883	465	182	136	100
Overweight										
5-14%	101.9	97.5	96.2	110.5	112.1	697	267	168	149	113
15-24%	97.3	93.9	103.7	106.4	75.5	355	116	113	90	36
25-34%	113.6	112.8	114.9	122.1	*	122	56	51	12	3
35-44%	92.3	81.7	*	*	*	18	12	4	--	2
45% and Over	*	*	*	*	*	1	1	--	--	--
SHORT HEIGHT										
Underweight										
25% or more	*	*	*	*	*	3	1	1	--	1
15-24%	134.6	153.8	133.7	141.6	*	54	27	13	10	4
5-14%	109.2	106.5	97.0	111.6	153.0	111	63	21	13	14
Average weight	95.6	98.0	65.3	106.1	122.4	94	55	12	14	13
Overweight										
5-14%	112.2	107.1	97.9	144.7	102.6	87	32	19	24	12
15-24%	118.1	98.2	159.2	73.9	139.1	52	14	22	7	9
25-34%	121.3	152.5	91.6	*	*	16	9	5	2	--
35-44%	*	*	*	*	*	2	2	--	--	--
45% and Over	*	*	*	*	*	--	--	--	--	--
MEDIUM HEIGHT										
Underweight										
25% or more	101.9	*	141.2	104.7	*	17	2	6	6	3
15-24%	96.2	108.9	85.1	82.2	97.0	222	107	50	36	29
5-14%	97.4	95.5	114.8	81.3	90.9	645	364	163	70	48
Average weight	98.2	96.7	96.4	98.4	109.3	669	342	147	101	79
Overweight										
5-14%	98.3	96.5	98.5	96.7	105.4	496	201	126	92	77
15-24%	96.7	97.2	98.8	110.7	66.7	260	91	76	69	24
25-34%	112.4	104.9	124.0	100.1	*	89	38	41	7	3
35-44%	91.1	82.9	*	*	*	13	9	2	--	2
45% and Over	*	*	*	*	*	1	1	--	--	--
TALL HEIGHT										
Underweight										
25% or more	*	*	*	*	*	3	1	1	--	1
15-24%	90.3	69.2	77.4	107.5	168.2	41	13	10	9	9
5-14%	109.5	122.3	98.9	109.0	64.6	135	78	30	20	7
Average weight	97.9	117.1	81.0	94.6	57.4	120	68	23	21	8
Overweight										
5-14%	111.5	95.3	84.1	142.4	150.0	114	34	23	33	24
15-24%	82.9	70.6	82.2	109.3	*	43	11	15	14	3
25-34%	112.7	119.7	85.3	*	*	17	9	5	3	--
35-44%	*	*	*	*	*	3	1	2	--	--
45% and Over	*	*	*	*	*	--	--	--	--	--

*Less than 5 deaths.

TABLE 4—Continued

Mortality of Women According to Build

Ratios of Actual to Expected Deaths by Metropolitan Female Mortality Table, 1922-1936 (Ungraduated). By Age Groups at Issue and by Duration of Insurance. Standard Issues Only.

Metropolitan Life Insurance Company, Ordinary Department, Issues of 1922-1934 Carried to Anniversary in 1936.

Build Group	SIXTH AND LATER POLICY YEARS									
	Per Cent. Actual of Expected Deaths					Deaths				
	All Ages	Under 30	30-39	40-49	50 & Over	All Ages	Under 30	30-39	40-49	50 & Over
GRAND TOTAL	100.0	100.0	100.0	100.0	100.0	3,314	1,356	848	680	430
Underweight										
25% or more	84.8	*	84.6	100.6	78.5	25	2	6	10	7
15-24%	91.9	100.0	95.3	81.0	83.4	294	114	81	60	39
5-14%	96.3	104.2	100.6	74.8	84.1	794	425	196	106	67
Average weight	102.8	98.6	99.4	127.9	87.0	893	386	201	208	98
Overweight										
5-14%	100.3	96.1	97.9	96.1	118.8	705	236	177	157	135
15-24%	102.4	100.2	92.8	107.5	115.7	416	121	107	117	71
25-34%	120.4	103.3	130.8	114.1	185.2	158	53	72	21	12
35-44%	94.8	84.4	110.5	*	*	26	16	8	1	1
45% and Over	*	*	*	*	*	3	3	--	--	--
SHORT HEIGHT										
Underweight										
25% or more	159.2	*	*	*	*	6	1	--	2	3
15-24%	104.9	93.2	149.1	73.3	108.2	41	14	14	6	7
5-14%	109.8	105.8	103.9	132.5	111.5	104	51	23	18	12
Average weight	104.2	97.2	99.3	160.1	70.8	96	45	18	24	9
Overweight										
5-14%	92.2	72.4	99.6	117.7	84.2	74	20	20	23	11
15-24%	128.7	142.2	106.4	122.4	154.9	68	21	17	16	14
25-34%	135.4	108.0	164.6	*	*	23	7	12	3	1
35-44%	*	*	*	*	*	3	2	1	--	--
45% and Over	*	*	*	*	*	1	1	--	--	--
MEDIUM HEIGHT										
Underweight										
25% or more	77.3	*	108.5	95.1	*	16	--	5	7	4
15-24%	88.5	102.9	78.9	79.4	85.8	206	85	48	44	29
5-14%	96.7	103.7	105.0	73.7	81.2	585	315	147	77	46
Average weight	102.4	96.7	103.4	125.6	86.9	667	284	159	152	72
Overweight										
5-14%	102.2	97.1	98.2	96.3	129.0	524	180	128	112	104
15-24%	98.4	93.6	91.2	105.4	108.6	290	85	73	83	49
25-34%	118.4	104.1	121.6	135.8	148.4	113	38	49	18	8
35-44%	104.5	92.3	135.1	*	*	21	13	7	1	--
45% and Over	*	*	*	*	*	2	2	--	--	--
TALL HEIGHT										
Underweight										
25% or more	*	*	*	*	*	3	1	1	1	--
15-24%	97.6	91.6	128.6	95.1	*	47	15	19	10	3
5-14%	84.3	105.5	79.4	46.5	73.5	105	59	26	11	9
Average weight	103.5	110.3	79.2	120.3	99.9	130	57	24	32	17
Overweight										
5-14%	96.9	110.4	95.6	80.1	100.3	107	36	29	22	20
15-24%	98.8	98.7	87.9	106.1	111.3	58	15	17	18	8
25-34%	117.1	96.4	147.3	*	*	22	8	11	--	3
35-44%	*	*	*	*	*	2	1	--	--	1
45% and Over	*	*	*	*	*	--	--	--	--	--

*Less than 5 deaths.

bined are of little import. Another important influence is medical selection, which is probably more severe where the weights are close to the limits, either underweight or overweight, permissible for Standard insurance. The irregularities in the mortality curves for the extreme weight groups are partly attributable to this factor.

Table 4 presents the ratios of actual to expected deaths at all ages and in four broad *age groups at issue* for the build groups described. The mortality ratios are computed on an ungraduated mortality table based upon the summed experience of these cases, by age at issue and by duration of insurance. The mortality ratios are also ungraduated, representing the actual mortalities recorded. Mortality ratios appear in the table for every group in which there were at least five actual deaths. This number is not, of course, large enough to give a reliable mortality ratio. It is necessary throughout in evaluating the ratios to keep in mind the number of deaths, which are also given in the table. Many of the irregularities in the mortality curves are due to the small size of the samples. Such irregularities are common in all mortality investigations. They occurred, for example, in the Joint Committee's investigation of the influence of build on the mortality of males, both in 1912 and in 1929. As Mr. Larus pointed out in his talk on Statistical Principles at last year's meeting, the amount of material necessary for an investigation which will show significant trends is surprisingly large. Ordinarily about 100 deaths are required if the material for investigation is fairly normal, although a smaller number is sufficient if the class is distinctly abnormal. It is too much to expect, in this or any mortality investigation, that the smaller sub-divisions will give an unmistakably regular trend. It is enough if a general pattern can be identified. The results of this investigation, in our judgment, satisfy this condition.

All Heights Combined

At *ages under 30,* the most favorable mortality (in the groups with sizable numbers of exposed to risk and deaths) was found among those slightly (5–14%) and moderately (15–24%) overweight, but the ratios for these groups were not significantly dif-

ferent from that for the average weights. From these low levels, the mortality increased to its highest point in the 25–34% overweight group. The next highest was for those 15–24% underweight. The limited experiences on the extreme overweights and underweights have been good. Despite a rather good progression of the mortality ratios from a high point at 15–24% underweight down to a low level among the moderate overweights and then up to another peak among the more obese young risks, the remarkable feature of the mortality at the younger ages has been the relatively small variation in the ratios according to weight, amounting to less than 12 per cent. if we except the highest overweight group which is based upon a relatively small experience. The mortality curves by weight in both the first five and sixth and later insurance years were essentially similar to those for the aggregate experience.

At *ages 30-39,* the situation was in many respects similar to that in the preceding age period. The lowest mortality ratio was found at 15–24% underweight, but since the ratios on either side of this group were considerably higher, it is apparent that underweight is still a slight disadvantage at these ages. The ratios in the average weight group and the first two overweight groups were slightly more favorable. The mortality then increased sharply to a maximum for the age period among those 25–34% overweight. It is interesting to note that apart from this one group, the range of mortality ratios at these ages also has been still fairly small—less than 15%.

Comparison of the results in the first five and the sixth and later years, showed a slight improvement in the relative mortality of the underweights with increasing duration of insurance, but the mortality of those 25–34% overweight rose sharply.

At *ages 40-49,* the best mortality was recorded among those slightly below average weight. The other underweight groups showed only slightly less favorable results. Curiously enough, the average weight group had a high mortality. We believe, however, that this finding is purely accidental. At any rate we have not been able to account for it. Apart from this anomaly, there was a steady upward progression of the mortality ratios with increasing

degree of overweight. In this age group, the relative mortality of underweights improved in the later insurance years. Consequently, the margin between the mortality ratios of the underweights and overweights was slightly higher in the later insurance years, as compared with the early years. Incidentally, it is at the longer durations that the mortality of the average weight group has been so bad. In the early insurance years, they showed an average mortality.

At *ages 50 and over,* the most advantageous mortality was among the underweights. The best showing was in the small experience on those 25% or more underweight. The mortality ratios rose steadily with increasing weight, except for a slight irregularity in the curve among the underweights and a more marked one among the overweights. But neither of these irregularities was contrary to the general trend of a favorable mortality among underweights and an excessive one among overweights. In the sixth and later insurance years, the trend was more regular than at all years combined. The advantage of the underweights and the increasing mortality of overweights then stood out more clearly. The picture in the early insurance years is somewhat confused, although, as a whole, the underweights had a better mortality than the overweights.

Mortality by Build in Separate Height Classes

The curves of mortality according to build in the several height classes have had the same general pattern, although there are numerous irregularities. These may arise from the small size of some of the groups, from the influence of medical selection or from other causes. At *ages under 30,* short and medium height women in the 15–24% underweight group had mortalities considerably above the general average, and the ratios decreased fairly regularly with increasing weight to a minimum among the overweights—in the 5–14% group among short women and in the 15–24% group among medium height women. Both curves rose sharply from their low point and the ratios at 25–34% overweight were at or near the peak for the age group. The amplitude of the mortality curve

of short women was much greater than in women of medium height.

The mortality curve according to build among tall women under 30 resembles those of the other two height groups in certain respects, namely, (1) a high mortality among those slightly underweight, as compared with those slightly or moderately overweight and (2) a steep rise in the mortality from the low point in the 15–24% overweight group to the 25–34% overweight group. The experience among tall women 15–24% underweight has been good, but the experience was not large. This departure from the general picture may not be of any great consequence. The size of the experiences on the short and tall women at ages under 30 was not large enough to yield significant information as to the effect of duration of insurance on the results. In the medium height group the build mortality curves of the two periods were roughly parallel.

The experiences at *ages 15-19 and 20-29* were analyzed separately for two weight groups, 15–24% underweight and 15–24% overweight, but the results were not significantly different from the aggregates for the two age groups.

At *ages 30-39,* the curves of mortality by build in the three height groups are strikingly similar. All begin at a high point on the underweight side. Among the short and tall women, the ratios dropped sharply from the initial point at 15–24% underweight to a minimum in the average weight group, and then rose steadily with increasing weight, except for a slight irregularity in the curve for tall women, to another peak at 25–34% overweight. Among the medium height women, the curve started at a high point at 25% or more underweight and dropped sharply to a minimum at 15–24% underweight, but then rose sharply again at 5–14% underweight. It declined gradually with increasing weight to a minimum at 15–24% overweight, and then increased abruptly at 25–34% overweight. Despite the seemingly irregularity of this curve, the general trend was a relatively high mortality among underweights, a low mortality in the average and moderately overweight women and a sharply increasing mortality among those excessively overweight.

At *ages 40-49,* there are points of similarity between the curves of the three height groups, but these are obscured by variations at certain points in each of the curves. The situation is best sum-

marized in the statement that in the aggregate the mortality is lowest among the underweights in each height group. In two of the height groups, this advantage of underweights was distinctly greater at the longer durations of insurance. The average weight women at 40-49 in all three height groups had a high mortality for which we have found no explanation. It may be noted, however, that in the medium height group the mortality ratio for average weight women is exceeded by that of the more obese and among tall women the differences between the ratios of the average and overweight groups are not significant.

At *ages 50 and over,* the data were much less abundant than at the younger ages and, consequently, there were considerable fluctuations in the curves of mortality by weight groups. Despite this, there was a clearly discernible trend in all height groups from a relatively low mortality among underweight women at these ages, to a high mortality among overweights. At the short and medium heights, this trend stood out even more clearly in the sixth and later insurance years than in the early years.

Discussion of the Mortality in Weight Groups

On the whole, the general picture of the mortality according to build stands out rather clearly. In its broader outlines, the picture is the same as among males, but with certain differences in detail which may be significant. The characteristics of the mortality curve vary with age. At the younger ages, the mortality is generally least favorable among underweights and those too much overweight. It is most favorable among those moderately overweight. Among women in their thirties, the extra mortality among the underweights begins to disappear; the point of most favorable mortality shifts toward the average weight group and the penalty of overweight becomes somewhat more pronounced. After 40, those moderately underweight do best, and overweight becomes an increasingly serious impairment. This trend becomes more definite with advancing age. These broad characteristics of the mortality by weight groups are not affected by height.

This general description fits both sexes. There may be, however, some difference in the degree of variation. We have made

a very rough comparison of the variations in the mortality ratios by age and weight groups with the similar data on males of the Medical Impairment Study. The ratios for females show less marked departures from the general average by weight groups. Thus, at ages under 30 the high mortality on the underweight side among males is 15% above average, compared with only 5% among our females. Likewise among men 25–34% overweight the excess is 34% compared to an excess of only 8% among our females. This phenomenon appears in every age group. It is not so clear among the overweights over age 50, but at this point our samples are relatively small.

Influence of Height on Mortality

In the aggregate, the mortality of women of short height has been distinctly above the average. The ratio of actual to expected deaths for this group was 109.0 per cent., compared with 98.8 per cent. recorded for both medium height and tall women. The high mortality of short women was found to exist both in the first five and the sixth and later insurance years. The mortality of medium height women was slightly better than that of tall women in the early insurance years, but this situation was reversed in the later years.

The short height women showed above-average mortalities in every age group and both in the early and late insurance years. The greatest excesses were found at the older ages. At 40-49, the mortality of short women was 121.1 per cent. or one-fifth worse than the general experience at these ages.

At ages under 30, the most favorable mortality has been recorded among medium height women. Between ages 30 and 39, the tall women have done best. Past 40 there is, in the aggregate, no marked difference between the mortality ratios of the medium height and tall women. There are, however, differences between the early and late insurance years in this respect. At the longer durations, the mortalities are decidedly most favorable among taller women. The facts on the influence of height on mortality are shown in Table 5.

TABLE 5

Influence of Height on Mortality of Women

Per Cent. Actual of Expected Deaths by Metropolitan Female Mortality Table, 1922-1936 (Ungraduated) By Height Groups and by Age Groups at Issue. Standard Issues Only. Metropolitan Life Insurance Company, Ordinary Department, Issues of 1922-1934 Carried to the Anniversary in 1936.

Age Height Group	Actual of Expected Deaths Per Cent.			Deaths		
	All years	First 5 years	6th and later years	All years	First 5 years	6th and later years
All Ages						
Short	109.0	109.8	108.2	835	419	416
Medium	98.8	98.1	99.6	4,836	2,412	2,424
Tall	98.8	102.0	95.8	950	476	474
Under 30						
Short	104.8	109.2	99.8	365	203	162
Medium	98.2	97.5	99.1	2,157	1,155	1,002
Tall	105.6	106.4	104.8	407	215	192
30–39						
Short	106.8	103.3	110.1	198	93	105
Medium	101.1	102.1	100.1	1,227	611	616
Tall	90.2	87.5	92.6	236	109	127
40–49						
Short	121.1	115.8	125.5	162	70	92
Medium	97.2	94.5	99.3	875	381	494
Tall	98.6	114.3	86.0	194	100	94
50 and over						
Short	111.1	116.9	106.2	110	53	57
Medium	98.9	97.4	100.3	577	265	312
Tall	96.2	99.0	94.0	113	52	61

We are not yet prepared to say what these variations in mortality according to height mean. It may be, of course, that the taller women are intrinsically better from the biological viewpoint. But we shall have to know more about the internal character of our group before we can be certain of such a conclusion. Indeed, we are inclined to believe that the favorable results in the tall group reflect racial and social factors more than biologic ones. For, we know that within the more favorably situated economic groups, the average height is greater than in the less favorably

situated groups, other things being equal. We know also that the shorter group includes a larger proportion than the tall group of foreign born women and women of foreign descent, among whom the economic and social status is, on the average, appreciably poorer than in native born women. These factors may be the cause of the differences shown, but a definite answer waits upon further investigation. Possibly a study of this material by amounts of insurance would help us to understand the differences and their causes.

Causes of Death

The mortality from certain causes of death varied greatly according to build. Some of the respiratory diseases took their heaviest toll among underweight women, while the cardiovascular and renal diseases, cancer, gall bladder conditions and diabetes were particularly prevalent among overweight women. Puerperal deaths also appeared high among overweights. Although this finding accords with clinical observation, this result is clouded because the rates are based on the total women exposed to risk and not on the number of births. Some causes of death show no distinct trend according to build. The relationship between causes of death and build are shown in Table 6.

This table gives the number of deaths from important causes in such detail as is warranted by the number of deaths, and ratios of actual to expected deaths from these causes have been computed on the basis of the death rates in the experience as a whole. Examination of the table shows that some of the variations in mortality according to build are little affected by age. In other cases, the differences diminish with advancing age, while in still others they are accentuated by age. In certain cases, the tendencies are accentuated with increasing duration of insurance when the effect of medical selection has worn off. Reference is made in the text to the findings according to duration when they are significant, but the figures have been omitted from the table.

The mortality from *tuberculosis* was 60 per cent. higher among those 15 per cent. or more underweight than the average for the group. The ratios declined progressively with increasing

TABLE 6

Mortality of Women from Specified Causes According to Build

Deaths and Ratios of Actual to Expected Deaths Calculated on Basis of Death Rates in Combined Standard Experience. By Build and Age Groups at Issue. Metropolitan Life Insurance Co., Ordinary Department, Issues of 1922-1934 Carried to Anniversary in 1936.

(Note: Ratios which apply to combined weight groups are centered between the limits of those groups.)

Cause of Death Age at Issue	Ratio Actual to Expected Deaths								Deaths							
	Underweight				Overweight				Underweight				Overweight			
	25% or more	15-24%	5-14%	Av. Wt.	5-14%	15-24%	25-34%	35-44%	25% or more	15-24%	5-14%	Av. Wt.	5-14%	15-24%	25-34%	35-44%
Influenza and Pneumonia, total																
All ages	192	131	91	101	101	85	97		8	66	123	141	111	52	23	
Under 30	142		79	87	125	116	116		29		56	58	51	23	13	
30-39	155		120	97	72	79	91		25		42	35	23	16	9	
40 and over	111		86	132	101	59			20		25	48	37	14		
Influenza																
All ages		165	91	92	112	89				26	39	40	38	22		
Under 40		173	105	86	96	91				20	37	29	23	17		
40 and over		67		110	123					8		11	20			
Broncho Pneumonia																
All ages		77		100	117	83				24		24	23	12		
Under 40		73		58	128	134				15		9	14	12		
Lobar and Unspecified Pneumonia																
All ages		119	99	108	89	76	134			31	69	77	50	24	17	
Under 30		89		99	123	112				43		35	27	19		
30-39		127		110	74					32		20	23			
40-49		109		141	64					18		18	14			
50 and over		103			101					11			8			
Tuberculosis, all forms																
All ages	160		134	96	73	33			134		378	260	127	42		
Under 30	140		132	96	76	38			91		322	221	104	36		
30-39	202		147	102	43				28		45	32	23			
40 and over	316		136		67				15		11		13			
Syphilis		109		111	91					12		9	12			

TABLE 6—Continued

Cancer, all forms																		
All ages	93	89	90	101	114	94	136		12	97	219	272	279	136	49			
Under 30		87	95	96	115	117	119			15	53	50	38	20	9			
30-39		100	90	99	101	107	134			33	63	72	66	45	24			
40-49		77	86	119	114	80	150			34	65	105	100	45	12			
50 and over		96	93	79	129	89				27	38	45	75	26	4			
Cancer of Stomach																		
All ages		127	84	93	115	84				15	21	27	31	16				
Under 40		172	65	94	116	81				7	7	10	10	6				
40 and over		103	99	93	114	86				8	14	17	21	10				
Cancer of Liver and Gall Bladder		106		128		89					16	15		17				
Cancer of Peritoneum, Intestines and Rectum																		
All ages		79		104	120	113					50	51	53	37				
Under 40		96		111	107	106					30	25	19	16				
40 and over		62		99	129	119					20	26	34	21				
Cancer of Genital Organs																		
All ages		99	89	92	110	105	142			32	63	72	79	45	16			
Under 30		105		104		101					17	12		13				
30-39		118	70	87	106	140	136			13	18	23	25	21	9			
40-49		81	102	88	116	106				9	22	22	29	17	3			
50 and over		91		99		95					16	15		23				
Cancer of Breast																		
All ages		93		106	113	99					67	58	56	37				
Under 30		119		81		118					18	9		15				
30-39		75		97	122	125					15	14	16	15				
40-49		90		131	94	84					25	28	20	13				
50 and over		99		91		113					9	7		14				
Cysts and Other Benign Tumors of Ovary		102		108		101					9	7		10				
Benign Tumors of Uterus		78		107	111	104					21	21	19	15				
Acute Rheumatic Fever		90		92	82						9	7	6					
Diabetes																		
All ages		105	69	68	122	162				11	18	20	30	27				
Under 40		102		77	67	155					20	12	7	12				
40 and over		54		57	163	168					9	8	23	15				
Pernicious Anemia		128		73		74					14	6		9				
Toxic Goiter		104		112		80					17	14		14				
Cardiovascular-renal Diseases																		
All ages		73	89	106	101	120	153	141		89	244	326	278	191	60	8		
Under 30		71	104	108	93	66	175	196		15	77	75	40	14	16	6		
30-39		47	91	94	104	141	156			14	63	67	67	58	28			
40-49		80	78	114	86	146	131			31	58	99	74	82	11			
50 and over		88	81	106	119	91	131			29	46	85	97	37	5			

TABLE 6—Continued

Cause of Death Age at Issue	Ratio Actual to Expected Deaths								Deaths							
	Underweight			Av. Wt.	Overweight				Underweight			Av. Wt.	Overweight			
	25% or more	15-24%	5-14%		5-14%	15-24%	25-34%	35-44%	25% or more	15-24%	5-14%		5-14%	15-24%	25-34%	35-44%
Cerebral Hemorrhage, Embolism, etc.																
All ages		81	82	117	96	106	174		3	23	48	80	62	40	13	
Under 40			98	114	92	144				1	20	23	15	20		
40-49	91		75	114	107	110				12	17	30	28	21		
50 and over	122		70	125	87	99				13	11	27	19	12		
Angina Pectoris and Coronary Artery Disease		75		103	120	143				19		21	24	20		
Organic Heart Disease																
All ages		77	91	105	100	114	149	186		44	122	153	126	83	30	6
Under 30		98	104	99	89	60	184			13	48	43	24	8	14	
30-39		71	83	96	111	132	124			11	30	36	37	28	13	
40-49		69	81	131	70	138	189			11	25	47	25	32	7	
50 and over		74	90	91	133	102				9	19	27	40	17		
Chronic Nephritis																
All ages		52	98	102	101	136	124			14	60	70	61	47	11	
Under 40		84		100	98	135	141			39		34	25	20	9	
40-49		68		108	64	201				14		17	10	24		
50 and over		100		100	135	49				21		19	26	5		
Acute Endocarditis and Myocarditis																
All ages		122		105		74				36		23		21		
Under 40		124		97		77				33		19		19		
Pleurisy		196			74					13			8			
Ulcer of Stomach and Duodenum		65		99		114				9		10		14		
Ulcer of Stomach		93		127		87				7		7		6		
Appendicitis																
All ages		90		88	121		118			60		44	44		31	
Under 40		93		83	121		130			51		34	33		26	
40 and over		79		110		104				9		10		16		
Intestinal Obstruction		67		115		118				15		19		23		
Cirrhosis of Liver		95		103		111				12		10		17		

TABLE 6—Continued

Biliary Calculi and Other Diseases of Gall Bladder, total																						
All ages			37			68	119	199		437			16		22	32	31		21			
Under 40			25			75	136	167		468			7		15	20	14		19			
40 and over			59			58	99		236				9		7	12		19				
Biliary Calculi			40			52	109	201		537			9		9	16	17		13			
Other Diseases of Gall Bladder			40			94	110		214				7		12	12		18				
Acute Nephritis			113			117		89					12		9		9					
Non-Venereal Diseases of Genital Organs, n. e. c.																						
All ages			96			108	92		109				35		29	19		18				
Under 40			101			114	77		102				32		26	13		14				
Diseases of the Puerperal State																						
All ages, 15-39	93		90	105	105		112		131		45		142	156	103		56		28			
15-29	92		87	104	105		125		159		33		111	121	74		42		22			
30-39	93		101	111	107			82			12		31	35	29			20				
Puerperal Septicemia																						
All ages, 15-39	91		87	101	106		111		175		14		45	50	34		18		12			
15-29		89		101	92			149				50		42	23			25				
30-39			90				122						17				16					
Puerperal Albuminuria																						
All ages, 15-39	137		83	96	110			124			13		26	28	21			17				
15-29	147		76	93	126			139			10		19	21	17			12				
30-39			109					85					17				9					
Puerperal Causes n. e. c. (except "other")																						
All ages, 15-39	77		95	114	100		104		116		18		70	78	46		25		12			
15-29	72		90	111	104			124			12		52	58	33			27				
30-39		106		123			86					24		20			23					
Suicide																						
All ages			101	101	111			86					54	41	34			19				
Under 40			95	97	119			102					39	30	25			16				
40 and over			123	115			75						15	11			12					
Accidents																						
All ages	96		102	87	109				104		29		92	79	72				51			
Under 30	98		102	95	97				107		15		60	53	32				24			
30-39		109		56	133				108			27		10	21				16			
40 and over		86		92	108				95			19		16	19				11			
Pedestrians in Automobile Accidents																						
All ages			63				147						13				18					
Under 40			64				149						9				11					

weight. In those 15 per cent. or more overweight, the mortality was only 33 per cent. of the expected. The differences were most marked in the age group 30 to 39 at issue. The mortality from *pneumonia and influenza* was likewise highest among those markedly underweight. But there was no significant difference between the other weight groups. At ages under 30, excessive mortalities were recorded both among those 15 per cent. or more underweight and among overweights. At ages 30 to 39, the mortality was excessive among underweights only. Past 40 the fluctuations were such as to obscure any relationship between build and mortality from these causes.

For *lobar pneumonia* (including pneumonia unspecified), the mortality was highest among underweights and those considerably overweight. This trend showed much variation with age. *Bronchopneumonia* caused a high mortality among young overweights. For *influenza,* the mortality was high, at all ages combined, among those 15–24 per cent. underweight. This was most noticeable at ages under 40, especially in the later insurance years. After 40 the death rate from influenza was highest among the overweights, but the excess was relatively small. The mortality from *pleurisy* among underweights was double that for the experience as a whole.

For the *chronic cardiovascular and renal conditions,* there was a fairly steady progression of the ratios from the lowest levels in the underweight groups to the highest in the overweight groups. This trend became more pronounced with increasing duration of insurance, but, surprisingly enough, showed up most clearly in the younger age groups at issue. This trend existed in each division of the cardiovascular and renal group, but was most marked for *organic heart disease. Acute heart and kidney disease,* on the other hand, tended to be high among underweights.

The mortality from *cancer* displayed a tendency to rise with increasing weight, although this trend was less marked than in the preceding groups. This relationship was most marked at the older ages, especially for *cancer of the female genital organs* and of the *intestines.* There was no distinct trend according to build for *cancer of the breast, stomach, liver* and *gall bladder.*

Diabetes showed the typical correlation between weight and mortality, far more distinctly than did most other causes. At all ages combined, the death rate among women 15 to 34 per cent. overweight was 62 per cent. above the average and among women 5 to 14 per cent. overweight, 22 per cent. above the average. The trend became clearer with increasing duration of insurance. The 15 to 34 per cent. overweight group had a mortality almost double the average in the sixth and later insurance years. These tendencies were more marked at ages past 40 than before that age.

The most striking differences in mortality according to build were found in *diseases of the gall bladder and biliary passages.* Here an unmistakable progression of mortality from a low point among underweights to a high point among more extreme overweights was disclosed. Women 25 to 44 per cent. overweight had a mortality from these conditions four and one-third times that of the average for the whole experience, and over ten times that of underweights. This trend was somewhat more distinct at the younger ages at issue. It was little affected by duration of insurance. It was somewhat more marked among cases with stones than among those with inflammatory conditions.

In the other surgical conditions, there was some variation in the picture. The mortality from *appendicitis* was slightly higher among overweights than among those of slighter build. There was no difference according to build in the mortality from *ovarian or uterine tumors,* nor from *other pelvic conditions.*

With regard to *puerperal conditions,* the results cannot be considered conclusive for reasons already pointed out. If, however, as we believe, there is no great difference in the proportion of married women in the various build groups and in their fertility, some weight may be given to the mortality from these conditions in the study. They show high death rates, particularly at ages under 30 among the more obese women. The mortality was higher than average in all the overweight groups at these ages. In the age group 30 to 39, in which the number of deaths from these conditions was much smaller, there was no distinct tendency according to build. With regard to specific conditions, *puerperal septicemia* followed the trend for puerperal conditions as a whole.

Puerperal albuminuria seemed to be high, both among the very thin women and among those much overweight. This was most marked at age groups under 30 at issue.

On the whole, the mortality from *accidents* did not show any great variation according to weight, although with increasing duration of insurance there seemed to be a tendency toward a high mortality among the more obese women. The mortality from accidents is relatively unimportant among women, and it did not seem worthwhile to make a detailed analysis according to the type of accident. For *pedestrian automobile accidents,* however, although the number was small, there was a much higher mortality among overweights than among those of slighter build. It is likely that this arises partly from the lack of agility and alertness on the part of stouter women.

Suicide showed no distinct variation according to build. Of the several minor causes studied, negative results, according to build, were obtained with regard to *syphilis, acute rheumatic fever* and *cirrhosis of the liver.* Underweights had a relatively high mortality from *pernicious anemia.* The mortality from *toxic goiter* was significantly below average among overweights, but there was no distinct difference between underweight and average weight women. *Ulcer of the stomach and duodenum,* as a group, had its lowest mortality among underweights, but this may reflect medical selection. Ulcer of the stomach separately was too infrequent to show any distinct tendency. *Intestinal obstruction* was low in underweights and somewhat high in average and overweight women.

Summary and Conclusions

1. The ungraduated data on build of women, as reported in 329,627 Standard policies issued by the Metropolitan Life Insurance Company, during 1922-1934 are presented in tabular form. These tables give the mean heights of these women at various ages and the mean and median weights and the standard deviations around the means at each inch of height from 4′8″ to 6′0″ in age classes. The total range of height was from 4′0″ to 6′6″. The

extremely short and tall women were excluded from the mortality study.

2. The mean heights increased steadily with age to a maximum between 40 and 49. The average height among adult women was approximately 5′4¼″. There is no evidence in this Standard material of an increase in the average height of women in the population in recent decades.

3. The average weights increased both with advancing age and increasing height. The increments of weight with height were fairly regular. On the other hand, the increments of weight with age were not regular. There was a definite tendency for a diminishing rate of increase with age during adult life and, past 50, the average weights became stable.

4. Appreciable differences exist between these averages and those of the standard (Medico-Actuarial) height-weight table. That table overstated the averages at ages under 25 and understated them at ages past 25.

5. Analysis of the mortality according to weight classes showed the same trends as the earlier studies on male lives, namely, relatively high mortalities among young underweights, distinctly high mortalities among older overweights and the most favorable build with regard to mortality shifting from moderate overweight at the younger ages to moderate underweight at the older ages. There is some evidence that the excess mortalities are not as great in females as in males.

6. The mortality of women of short height has been distinctly above the average. In general, tall women have had the best mortality except at ages under 30. The biological significance of this finding is questionable. It may be related to social and racial factors and may be temporary.

7. Analysis of the mortality from specific causes of death, according to build, revealed certain definite characteristics. Most of the respiratory diseases were excessively frequent among younger women markedly underweight, but obese women also suffered high death rates from some of these conditions. Among overweight women high death rates prevailed for all of the cardiovascular-renal conditions, diabetes, gall bladder disease, cancer

and puerperal conditions. Generally, the greater the degree of overweight, the higher was the mortality from these diseases. The mortality from accidental causes, as a whole, and from suicide showed no marked variation in mortality according to weight.

This study of our experience on women has yielded important new anthropometric and mortality data. It must be emphasized that this is a preliminary report, based, as it is, on Standard issues only. Some of our findings may be modified when the Substandard data, which we are now analyzing, are ready. In the main, however, the general picture given by these Standard risks is a consistent one.

Our anthropometric data, we believe, indicate the need of some revision of the standard height-weight table. That table is too smooth and its very smoothness disregards what are probably fundamental changes in weight according to age during adult life. A complete statement in this regard must wait until we have the facts for the Substandard because this group contains the data on the extreme underweights and overweights. Incidentally, it is our judgment that median weights are probably better than mean weights because the latter are unduly influenced by the extreme overweight cases.

This mortality experience confirms generally the assumption that has been made in the insurance business with regard to build of women, namely, that they present a situation similar to that disclosed by the studies of the influence of build on mortality among men. There is some evidence, however, that the penalties that go with underweight at the younger ages and overweight at the older ages are not as severe as in men. This conclusion, however, is stated only tentatively. We must have the results on our Substandard issues before we can be certain of this conclusion. Medical selection of the overweights given Standard insurance may account for the relatively favorable mortality of female overweights as compared with males.

DR. BROWN—Very appropriately one of the authors of this paper suggested that an anthropologist be invited to discuss it, and I feel that we are fortunate in the selection which has been

made. I learn that the person whom I am about to introduce was a brilliant student and a magna cum laude in anthropology when he graduated from Harvard in 1923.

Many of us took great pleasure in the stories "Mutiny on the Bounty," "Men Against the Sea," and "Pitcairn Island," and so I think that if for no other reason we should have a warm spot in our hearts for one who created the volume entitled "The Heritage of the Bounty: The Story of Pitcairn Through Six Generations." The Alaskan Eskimo, Society Islanders, Cook Islanders and the Javanese, and their physical characteristics, have been the subjects of brilliant work by him. He is Associate Curator of the American Museum of Natural History, New York City. It is with great pleasure that I introduce Dr. Harry Lionel Shapiro, who will discuss the paper which is now before the meeting.

Dr. Shapiro—After Dr. Dublin's admirable paper I am afraid the few sheaves I am able to bring forward will appear very meagre, indeed. For the good harvester leaves little for the gleaner.

Before, however, discharging the pleasant task of discussing some of the implications of Dr. Dublin's paper, I cannot forbear digressing for a moment to touch upon its methodology. On reading the copy of his paper which Dr. Dublin sent me, I was struck by the paradox of one of our most accomplished statisticians using the simplest kind of statistical tools to draw from a Goliath of a series the most conservative of conclusions. On the contrary, we physical anthropologists, or at least our "emancipated" younger generation, wrestling with pygmy series, resort to the most complicated statistical toe-holds and half-nelsons only to end up frequently by being floored by our own data. Perhaps there is a moral for us in this antithesis which may be stated in paraphrase. Physical anthropologists rush in where statisticians fear to tread.

The subject of Dr. Dublin's paper especially pleased me because I see it as a step in a significant direction. I may perhaps best explain this by a bit of personalia. Last spring, the Milbank Fund held a roundtable meeting in order to discuss and, if possible, to clarify the criteria by which quality in human populations might

be appraised. The conference was designed to consider three approaches to the problem: the genetic, the psychological and the physical anthropological; the last to deal with the purely physical criteria of quality. I was entrusted with the responsibility of supplying the anthropological contribution to the banquet. When, however, I repaired to the anthropological cupboard, I discovered that the large canister, cheerfully labelled "Human populations, quality of," was quite empty. By that time, with the imminent arrival of the guests, it was much too late to run down to the corner grocery.

The survey which I made, at that time, of our anthropological measures of physical quality revealed to me the poverty of our knowledge on this vital question. It also brought home the fact that we had been talking glibly for a long time about something we had never bothered to define rigidly and precisely. Physical quality is one of those general ideas upon which everyone seems ready to discourse and which appears to be self-evident. In reality, however, the common notion of physical quality is adulterated with all kinds of emotional, esthetic and traditional misconceptions. The literature is full of warnings of the physical deterioration of our population.

Now all this pessimism may be quite justified and we may be facing a lamentable condition requiring prompt action, but the number of convincing, careful studies on physical quality in populations are extremely limited. In my Milbank paper, I made a plea for just such studies on groups of people. I attempted to define physical quality as the combination of functional efficiency and of survival value. But this is far from being an absolute definition, for judgment of quality is conditioned by purpose. What constitutes efficiency for one purpose may be detrimental in another context. And even survival value in terms of longevity serves no useful function for the race if carried to excess.

But even though quality judgments may be merely relative and applicable only to individuals, never absolute and never attributable to whole groups, nevertheless we have reached a stage in the study of human populations where it is essential to know the specific characteristics of our population in terms of specific

efficiencies and survival values. Intelligent understanding of our population and far-sighted care for its future welfare demand exact, reliable information of this nature.

Dr. Dublin's paper fulfills these requirements. He has investigated specifically the mortality rate of an enormous number of women divided according to easily determined types of body-build, and he has demonstrated within the limits of his data a pattern of mortality associated with various degrees of under and over weight, age and stature. Here is concrete material, available for criticism, acceptance or modification. It is above all tangible evidence from which conclusions may be drawn and benefits derived. We need more of such objective studies which serve to link physical criteria with function and survival.

Lest Dr. Dublin be falsely accused of importing a quite unnecessary *claqueur,* I hasten now to demonstrate his purity by offering a few mild reservations and suggestions. I gather from Dr. Dublin's remarks that the recorded weights and heights employed in his study represent these characteristics of the subject at the time of the issue of the insurance policy and not at any intervening period before death. This assumes that body-build, as determined from a single record, continues as a stable entity. Now, it seems to be true that some individuals maintain fairly constant height-weight ratios over considerable periods of time, but we are all familiar with the occurrence of rapid changes in other subjects, especially in weight and especially at certain times of life. Is it not conceivable, therefore, that the height and weight at the issuance of a policy are not sufficiently diagnostic? I cannot, of course, predict whether or not this is a point of any moment in the analysis of the present data which has a different orientation, but it does suggest itself as worthy of investigation.

Another question which rises to my mind is the matter of the determination of body-build. According to anthropological criteria, height and weight alone, useful as they are for a rough and ready classification, are not adequate as an index of body-build. Individuals of similar heights and weights may, and often do, differ to a large degree in their body structure and even in the proportion of fatty tissue. The student of constitution con-

stantly meets with wide variation within such coarse categories. It occurs to me that possibly the irregularities of Dr. Dublin's data may find a partial explanation in just such factors as this.

I find also I must dissent on the question of increasing height. It is true that in the mean statures for the various age groups of the present data there is no evidence of greater height in the younger age groups, but rather the contrary; at least the maximum appears in the 45-49 year groups. Dr. Dublin suggests that the absence of a maximum in the younger groups may be in part the consequence of a greater increase in recent years of policies issued to the shorter foreign-born women, but he seems to prefer the conclusion that no increment has actually occurred. In opposition to these findings, I must cite the work of Bowles on American college women, the studies of Boas on second and third generation immigrants. The data of Hrdlicka on old Americans and the series of a large number of European and Japanese workers all agree in demonstrating a progressive increase in stature by generations. In the case of the measurements from the canton of Ste. Marie Vesubie, the increase has been progressive for over a century. These studies are too overwhelming to be denied. I should like to intimate that it may be more reasonable to give greater emphasis to Dr. Dublin's own explanation of a change in racial composition and also to offer another. The increments which occur appear from Bowles' work to be slight—about 1 cm. or about 4/10 of an inch every 12½ years. Furthermore, individual stature often continues to increase until well into maturity. Therefore, all these—the change in the racial origins of the applicants, the size of the increment to be expected within a decade, and the continued growth of the individual—may well account for the absence of any evidence of stature increase in the present data.

Dr. Dublin mentioned that a large number of his subjects were described in height and weight not by actual measurement but by the subject's own statement. Dr. Dublin, of course, was thoroughly well aware of the disadvantages of such information, but, under the circumstances, he has had to make use of what material was available. I am inclined to agree with his assumption that these estimates were approximately correct and, in their

aggregate form, close to the truth. But would it not be possible to check the accuracy of a subject's statement against the actual measurements in a small but statistically adequate sample in order to obtain some idea of the general reliability of such estimates as these? The slight investment of time which this involves would be amply repaid in the enhanced reliability of vast stores of data in the files of insurance companies.

It is a well-known truism that research propounds more questions than it answers. This study, likewise, opens up many inviting leads and stimulates the imagination. It would be easy to launch forth on the possibilities for further research which this investigation suggests, but to do that would lead me into pastures not assigned to me. I should like, therefore, to close these inadequate comments with a reiteration of the great value which researches such as Dr. Dublin's have for our understanding of the biological behavior and characteristics of human populations. No one, I believe, would have predicted in the infancy of the life insurance business that a new and extremely powerful agency for research was taking form. Yet, partly as a by-product of more specialized inquiries necessary to the efficient conduct of their affairs, the various companies have collected data of incalculable value to students of populations. They have amassed information which no other group could have obtained. I should like to see this function extended, if possible, with the co-operation of physical anthropologists. Such an alliance could yield results of mutual benefit.

Dr. Brown—I ask Dr. Richard L. Willis, of the Mutual Life of New York, to continue with the discussion.

Dr. Willis—When I read this paper by Dr. Dublin and Mr. Marks for the first time, I decided to make notations on the margin of any questions which came to my mind. I was surprised when I finished the paper to find that every question had been answered in the paragraphs immediately following. I feel that, where a writer has anticipated and answered every question of

the reader, that writer has written a really good paper—certainly in the mind of that particular reader.

In earlier reports on the mortality of women, it has been shown that women are better risks than men. This paper, I feel, gives one of the reasons why this is true. This report shows that, in women between 5 feet, 1 inch and 6 feet tall, the mortality is little affected by variations from the mean or average in weight, that is, variations from 25% underweight to 25% overweight. The mortality of women between the heights mentioned is decidedly better than in men. The findings show what probably was to be expected on account of earlier similar findings in men; that is, that the mortality is higher among light-weights under 30 and heavy-weights over 40 years of age. This mortality also is considerably less than the mortality of men in similar groups.

The study also shows that short women, that is, women between 4 feet, 8 inches and 5 feet, 1 inch in height, are not as good life insurance risks as taller women. Most of us have felt for a long time that child-bearing was an added hazard in these very short women. This study shows that the mortality is higher, not only in the younger ages, but also in women beyond the child-bearing age, that is, in the groups between 40 and 49 years of age, and in those over 50 years of age. The evidence seems very conclusive, with 835 deaths in this group, that these short women are not as good risks as the taller women. However, the vast majority of risks, over 85%, are included in the groups between 5 feet, 1 inch and 6 feet, and these are all good except in the young extreme light-weights and the older excessive over-weights.

The gradual increase with age in the weight of women of all heights up to age 50 seems to be physiological and, therefore, should *not* affect the mortality. The great desire of women during the past several years for "stream-lined" figures apparently has not affected the average or mean weight in any of the age groups.

This study was made on four age groups; namely, those under 30, those from 30 to 39, those from 40 to 49 and those over age 50. By far, the largest group is contained in those under age 30, which had 2,300 of the 6,600 deaths reported. It looks as though some

advantage might have been gained if this group had been subdivided, as the men's study was, into those from 15 to 19, and 20 to 29, or even from 10 to 19, and 20 to 29 years of age. Perhaps this has already been done without showing significant differences in the mortality findings of the two groups. Dr. Dublin can tell us about this. I think that the authors are much too conservative in their estimate of the value of these findings without further study. It seems to me that the evidence is ample to show that the penalties which go with under-weights in the younger ages and over-weights in the older ages should *not* be as severe as in men, and perhaps there should be *no* penalty except ... extreme cases.

I do not wish to detract from the medical selection as practiced by the doctors of the Metropolitan Staff, but I do not feel that their selection accounts for the relatively favorable mortality of female over-weights as compared with males. I presume that the same doctors selected the males. I believe that the reason for this favorable mortality in all the groups of women over 5 feet, 1 inch tall is better explained by "something inherent" in themselves. Just what that something is would make an excellent study for a future paper to be presented to this Association.

This Association is greatly indebted to Dr. Dublin and Mr. Marks, who, by this excellent paper, have given us new and important proof that women are better life insurance risks than men.

DR. BROWN—Mr. Marks, will you close the discussion?

MR. MARKS—There is very little to add, but I should like to address myself to a few of the comments made by Dr. Shapiro.

First, the height-weight ratio does change with age, but I doubt if there is much shifting of individuals from the underweight group to the overweight group, that is, in the underweight group at age 25 are most of those people who are going to be underweight at age 50, and in the average weight group, at 25, are the great majority of the future average weight group at age 50.

These changes, however, have little direct interest to us because we are chiefly concerned with what is going to happen to people in groups classified as of the time of examination.

With regard to the change or lack of change in the average height, we are in pretty good agreement. Our paper has made clear that probably the fundamental reason why we do not find any increase in the average height with the passing of time is that our group is not homogeneous. Most of the anthropological literature relates to much more homogeneous groups.

As Dr. Dublin pointed out, although he preferred not to stress it, there is some increase in the average height at the younger ages. It is just beginning to show up. That may be the crucial point of the matter because the younger people among our later issues, who have a somewhat greater average height than the younger people represented by our issues 15 years ago, are almost all native born, and that is not true of those insured at these ages 15 years ago. The restriction of immigration into this country is largely responsible for the change, at ages 15 to 25, in the percentage of the native born. That represents a new and definite influence, and probably ten years from now our insurance figures will begin to show more distinctly and over a greater range of life what the anthropological data showed; namely, that the average height has definitely increased.

With regard to increasing height with age, Dr. Shapiro pointed out that the maximum height is usually reached in maturity. We recently reviewed a good deal of material in the literature that brought that out. Such studies go back as far as 1870. There is one point that we should not overlook with regard to this matter; namely, that the increasing average height with age up to maturity, may represent not an actual increase in the average height, but rather a differential survivorship between shorter and taller individuals. Most of the studies to which Dr. Shapiro and I referred relate not to the same individuals studied periodically over a number of years, but to aggregates of a population classified according to age. Those aggregates so classified may themselves, therefore, conceal a differential mortality according to height, and

not necessarily prove a longer period of growth than is ordinarily assumed to exist.

* * * * * *

Dr. Brown—Dr. H. B. Anderson, of the Imperial Life Assurance Company of Canada, has some facts to communicate to us concerning poliomyelitis.

THE CONSIDERATION OF A POSSIBLE ROLE OF WINDBORNE (HAYFEVER) POLLENS IN THE DISSEMINATION OF THE VIRUS OF POLIOMYELITIS

H. B. Anderson, M. D., F. R. C. P. (C)

Medical Director, Imperial Life Assurance Company, Toronto

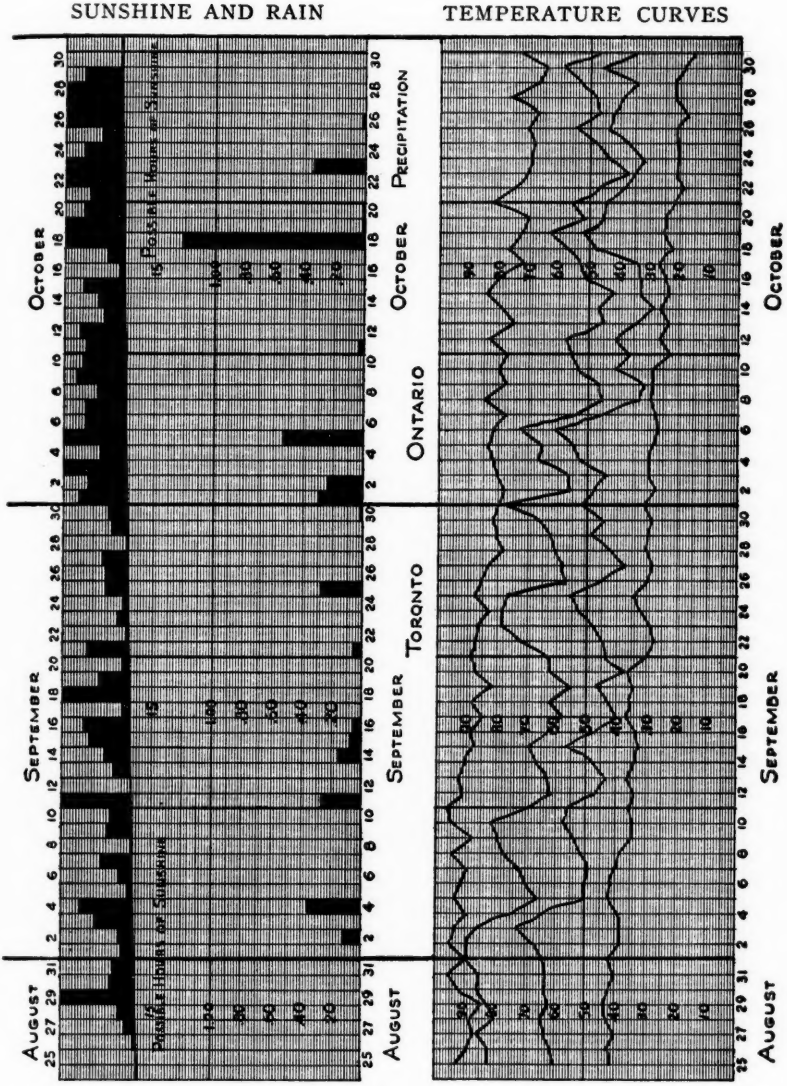
In July last, an unusually prolific and widespread ragweed infestation of Toronto, its suburbs and throughout the Province of Ontario occasioned a large correspondence, in which I was concerned, in the city and provincial daily press, with numerous editorials, urging the necessity for prompt and vigorous action toward its control.

In the midst of this newspaper publicity an explosive outbreak of poliomyelitis began and the coincidence of ragweed infestation and the epidemic caused me to wonder if there was not a possible aetiological relationship between the two conditions. Having this possibility in mind, I watched the course of the epidemic and have formulated my observations and data as follows.

Outbreaks of clinical poliomyelitis and aestivo-autumnal hayfever have approximately the same seasonal incidence. "Both in endemic and epidemic prevalence, poliomyelitis is characteristically a disease of summer and early fall." (Report of the International Committee for the Study of Infantile Paralysis.) The seasonal coincidence is recurrent with all major epidemics of poliomyelitis.

The sudden decline of cases both of hayfever and poliomyelitis following the onset of autumn frosts is even more striking. That this phenomenon, in so far as poliomyelitis is concerned, is not due to cold *per se* appears probable because the virus is cold resistant and moreover winter epidemics occasionally occur.

While mature pollens also are resistant to frost, the effect of cold, rain and winds upon ragweed and other hayfever-producing weeds interferes with pollination, and consequently pollen-contamination of the air drops suddenly. This occurred during the



BLACK BARS, left side of graph, indicate possible hours of sunshine.

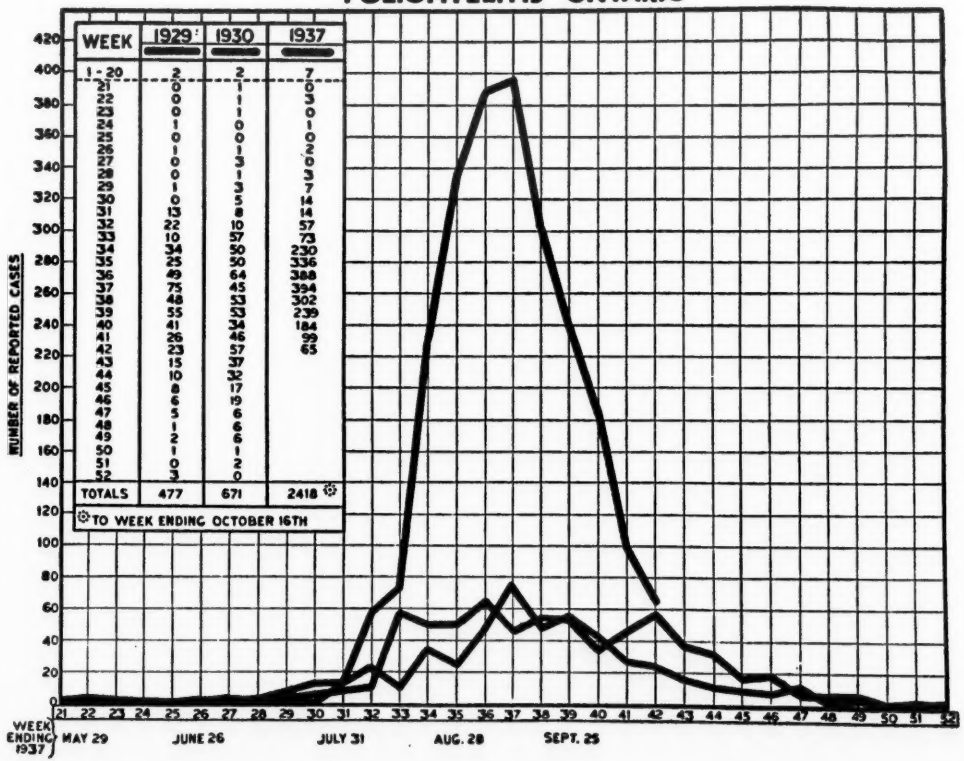
LIGHT BARRED AREAS indicate daily sunshine.

BLACK BARS, right side, indicate rain.

Left and right, indicate maximum and minimum temperatures since 1840.

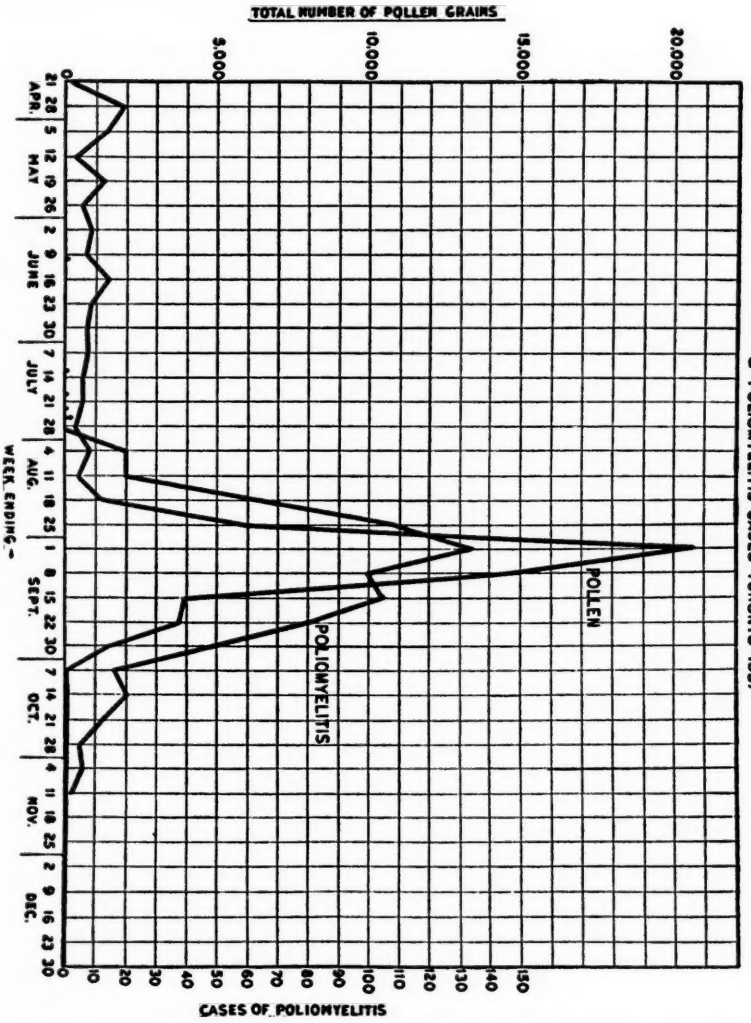
Central curves, indicate maximum and minimum daily temperatures for corresponding period during 1937.

POLIOMYELITIS - ONTARIO



WEEK	1929	1930	1937
1 - 20	2	2	7
21	0	1	0
22	0	1	3
23	0	1	0
24	1	0	1
25	0	0	0
26	1	1	2
27	0	3	0
28	0	1	3
29	1	3	7
30	0	5	14
31	13	8	14
32	22	10	57
33	10	57	73
34	34	50	230
35	25	50	336
36	49	64	388
37	75	45	394
38	48	53	302
39	55	53	239
40	41	34	184
41	26	46	99
42	23	57	65
43	15	37	
44	10	32	
45	8	17	
46	6	19	
47	5	6	
48	1	6	
49	2	6	
50	1	1	
51	0	2	
52	3	0	
TOTALS	477	671	2418 ⚙

⚙ TO WEEK ENDING OCTOBER 16TH



POLLEN CONTENT OF THE AIR-TORONTO-1932
& POLIOMYELITIS CASES-TORONTO-1937

The pollen content of the air indicated by graph, is for 1932, as counts for 1937 were not available. As pollen counts show marked yearly and even daily variation, due to weather, wind-direction, location and other factors, it is evident that, apart from suggesting a roughly corresponding seasonal co-incidence with poliomyelitis, no precise scientific significance can be attached to it.

Pollen counts are an indefinite measure of pollen contamination of the air on the dates when counts are made, whereas cases of poliomyelitis are published as week-end reports. To estimate infection-dates, allowance should be made for the period of incubation and delays in diagnosis and reporting. These factors represent an indefinite time varying from 10 days to a fortnight, more or less. Thus a case of poliomyelitis recorded on August 28th, would probably indicate infection between August 14th and August 18th.

recent Ontario epidemic which subsided some weeks earlier than was anticipated.

In Ontario and the North-eastern United States, the geographical distribution of clinical poliomyelitis is roughly that of abundant infestation of hayfever-producing weeds. Both conditions are most widespread in Northern latitudes and both are uncommon in tropical countries, where insect rather than windborne pollination predominates.

With the increasing incidence of poliomyelitis in the past forty years there has been an increasing infestation of hayfever-producing weeds, due to motor traffic, well exemplified by the almost hedge-like distribution of ragweed along the sides of busy highways and neglected boulevards whence seeds are scattered by traffic and carried on cars to various destinations. It has been noted by Wickman and others that poliomyelitis follows lines of communication.

The human nasopharynx is the reservoir of the virus of poliomyelitis, the path of entry and "the path of elimination of the virus." The virus is widespread, of high infectivity but of low virulence, only about one in a hundred of those infected developing the clinical disease. The spread of the virus, according to prevailing opinion, is by carriers either through direct or indirect contact or by means of droplet infection.

W. F. and Mildred Weeks Wells and their co-workers, in the past few years, have demonstrated droplet nuclei, the dried residues of small droplets less than a tenth millimeter in diameter, capable of carrying infection and of floating and drifting in the air for periods which permit its dissemination. They have also demonstrated the effect of expulsive expiratory efforts, especially sneezing, in enormously increasing bacterial contamination of the air of rooms and semi-enclosed spaces. "Whenever a person sneezes many thousand nasopharyngeal organisms remain suspended in the air and in commonly occupied enclosed spaces the exchange of nasopharyngeal flora is inevitable."

Hayfever pollens induce coughing and sneezing in between two and three millions of the population of the North American continent, many of whom presumably are 'carriers' of the virus, who,

by this means, evacuate the contents of their nasopharyngeal cavities, thus contaminating the air with droplets and droplet nuclei from the reservoirs of the virus of poliomyelitis. Since sneezing and coughing are not symptoms of poliomyelitis in children nor do children as a rule suffer from hayfever, it would seem that a logical means of spread would be the inducing by pollens of sneezing and coughing in adult virus carriers.

In this paper no addition to the known epidemiology of poliomyelitis is implied other than to suggest a mechanism by which the nasopharyngeal secretions, containing the virus, are disseminated.

NOTE—This is a preliminary communication; an abstract of a paper given later before the Academy of Medicine, Toronto, on December 10, 1937.

DR. BROWN—Dr. Anderson, I thank you for having brought these things to our attention. I feel that the Association should feel honored that you would come to us to speak on this subject. Your thoughts are of very great interest to us.

* * * * * *

DR. BROWN—Our friends of the Sun Life have made some studies concerning diabetic family histories. This work was recently completed and was not announced in our printed program. Dr. Birchard will speak to us.

INHERITANCE OF DIABETES (STATISTICAL STUDY OF A SMALL GROUP WITH FAMILY HISTORIES OF DIABETES)

J. K. Gordon, M. D., C. M., F. R. C. P. (C)
C. C. Birchard, M. D., M. R. C. P. (L), F. R. C. P. (C)
Sun Life Assurance Company of Canada

In the autumn of 1936 the coincidental appearance of a number of medical reports where glycosurias on examination and family histories of diabetes appeared in the same applicants suggested to one of us (Gordon) that we might investigate with profit the corelationship of such family histories and diabetes—the statistical experiment to consist of determining the incidence of glycosuria in such applicants by sugar tolerance tests—full carbohydrate meals plus examinations of urine only or, where practicable, glucose test meals plus examinations of bloods and urines. This experiment was started in the autumn of 1936 and has been continued since then with results which are sufficiently striking that it appears to us advisable that they should be put on record in the transactions of the 1937 meeting of this Association. Accordingly, the data is offered to the executive of the Association, but as a preliminary report only.

In the period mentioned we have had some 225 odd white lives submitted in Canada and U. S. A. which were *essentially unblemished except for history of diabetes in parents or siblings.* Seven only gave histories of diabetes in a parent and one sibling. Two cases had two siblings affected. Of these 225 odd cases we asked for sugar tolerance tests in 208. In a number of cases the test was not demanded on account of our encountering some degree of resistance on the part of the Company's agency forces and it did not appear to be expedient to be unyielding or arbitrary. But this should not vitiate the status of the group as a very small random sample of the insuring population of Canada and U. S. A. Of the 208 cases from whom special tests were asked, we were able to complete these on 182 and the results are set out in the five tabulations which follow:

RESULT OF INVESTIGATION OF DIABETIC FAMILY HISTORY

TABLE 1

(All cases investigated)

Number of applications with diabetic family history in which test meals and sugar tolerance curves were completed in Canada and the United States from September, 1936, to September, 1937.

	Tests Requested No.	Tests Completed No.	Tests Completed Amount	Found Positive No.	Found Positive Amount	Rated No.	Rated Amount	Postponed or Declined No.	Postponed or Declined Amount	% Postponed, Rated or Declined No.	% Postponed, Rated or Declined Amount
Canada	67	65	$133,500	7	$18,000	2	$ 8,000	5	$10,000	10.8%	13.4%
United States	141	117	575,200	7	40,500	3	30,000	4	10,500	6.6%	7.1%
TOTAL	208	182	$708,700	14	$58,500	5	$38,000	9	$20,500	7.7%±1.3%	8.3%±1.4%

TABLE 2

(Cases investigated by glucose meals, bloods and urines examined)

	Number	Found Positive
Canada	1	1
United States	3	1
TOTAL	4	2

TABLE 3

(All cases—Family History)

	Tests Completed No.	Tests Positive No.	Tests Positive %
One parent diabetic	145	11	7.6%
Two parents diabetic	....	....	.....
One sibling	28	2	7.1%
Two siblings or more	2	1	50.0%
One parent and one sibling	7	....	.....
TOTAL	182	14	7.7%

TABLE 4

(All cases—sex)

	Tests Completed No.	Tests Positive No.	Tests Positive %
Male lives	161	13	8.7%
Female lives	21	2	9.5%
TOTAL	182	14	

TABLE 5

(All cases—Age grouping)

Ages	Tests Completed No.	Tests Positive No.	Tests Positive %
–30	62	4	6.4%
31–49	101	8	7.9%
50	19	2	10.5%
TOTAL	182		

The special tests consisted of: (a) A meal rich in carbohydrate, the urine being collected at the three hour period, or (b) fifty grammes of glucose, bloods and urines being examined in the fasting state, and at the half hour, one and a half hour, and two and a half hour periods. Of the 182 tests completed, 178 were by the former technique and four by the latter.

Where the test consisted of carbohydrate meal and urines only examined, we took as a positive result percentages of 0.25 or more of sugar and the percentages ranged all the way from 0.25% to 1.6% and the average of the group was 0.52%.

The important result in our opinions is that 7.5%, ± 1.3 by lives, and 8%, ± 1.4 by amounts, of persons unimpaired except for family histories of diabetes should by submission to special tests have shown more or less impairment of carbohydrate tolerance, some frankly diabetic, and all to a degree that for insurance purpose they had to be declined, rated or postponed as insurance risks.

One would like to add the further comment that it should be kept in mind that we had no very satisfactory authentication of the character of the carbohydrate meal taken and the identity of the urine specimens in a proportion of those cases where the special tests consisted of a carbohydrate meal and urines only. We believe that, if we had had in all cases proper glucose test meals with bloods and urines examined, a larger proportion of these lives would have been transferred from the standard to substandard and uninsurable groups. Another point worthy of note is that the average age of the 182 lives submitted to test was only 35.5 years and that 163 were aged 49 years or under. Since it is a well recognized fact that persons carrying the hereditary diabetic taint are more likely to exhibit the disease as their ages increase, one may safely draw the conclusion that similar investigation on older lives would show a corresponding increase in the incidence of diabetes.

It is appreciated by the authors that our group is small and the results are by no means conclusive. Our reason in making a report at this time is that we hope that this contribution may

stimulate other life offices to conduct similar investigations during the ensuing year.
OCTOBER, 1937.

* * * * * *

At this point, through the courtesy of Dr. William B. Kountz, of Washington University, St. Louis, and the kindness of Dr. Harry E. Ungerleider, of the Equitable Life Assurance Society, a film was shown entitled "Revival of the Human Heart." Dr. Ungerleider explained that the film illustrated an experiment in physiology, having no relation to mortality or underwriting.

* * * * * *

DR. BROWN—The electrocardiogram established itself as a part of the armamentarium of life insurance medical selection very early in this decade. Electrocardiologists appeared with a suddenness and in numbers comparable to the advent of mushrooms on a dewy lawn on a September morn. But I think the membership of this Association realized very early the truth of an old adage, "A little knowledge is a dangerous thing." In this membership there are men whose electrocardiographic knowledge and judgment stand shoulder to shoulder with the best. It is to those that the companies have looked for advice and decision, but our knowledge is not complete—possibly only a beginning has been made.

The Professor of Internal Medicine at the University of Michigan primarily interested in this subject, and one of the leaders throughout the world in the development of fundamental information and current advances in that knowledge, endeavoring to apply that information to the practical problems, has very kindly come to us today. Dr. Frank N. Wilson has chosen as the subject of his discourse, "Recent Advances in Electrocardiography."

RECENT PROGRESS IN ELECTROCARDIOGRAPHY AND THE INTERPRETATION OF BORDERLINE ELECTROCARDIOGRAMS*

FRANK N. WILSON, M. D.

Professor of Medicine, University of Michigan

I appreciate the courtesy shown me in inviting me to come here to talk to you about the electrocardiogram. I presume that I was invited for several different reasons.

About a year ago, I tried to convince your President, Dr. Brown, that the life insurance companies could make a most important contribution to our knowledge of the electrocardiogram if they would undertake a comprehensive statistical study of the electrocardiograms of a large number of normal subjects of both sexes and of all ages. He seemed rather skeptical, but my presence here is no doubt due to a desire on his part to give me an opportunity to say a few words on this subject to a large audience.

It is difficult for those of us who are using the electrocardiograph in a hospital to obtain the material necessary for an adequate study of the normal electrocardiogram. Almost all of those whom we have occasion to examine either have heart disease or are suspected of having it, and we have no easy way of obtaining for electrocardiographic examination the large number of normal subjects required for a study of the kind to which I have referred.

On the other hand, applicants for life insurance are, for the most part, in good health and represent a fair cross-section of the general population. Only the life insurance companies have the

*From the Department of Internal Medicine, University of Michigan Medical School. The studies described in this dissertation were carried out with the help of a grant made to Frank N. Wilson by the Horace H. Rackham School of Graduate Studies.

Some of the illustrations which appear on the following pages (Figs. 1, 6, 7, 8) were first published in an article by Wilson, which appeared in "Diseases of the Coronary Arteries and Cardiac Pain", edited by Levy and published by The Macmillan Company; others, (Figs. 2, 3, 10, 11, 12, 13, 14, 15) were first published in an article by Wilson, which appeared in "The Piersol Cyclopedia of Medicine" published by F. A. Davis Company. Permission to reproduce these illustrations here has kindly been granted by these companies.

necessary material and facilities for making a study of the normal electrocardiogram that would enable us to determine with the desired accuracy the probability that a given electrocardiogram is within or outside the normal range of variation.

The life insurance companies can also help in the solution of other closely related problems. They can more readily determine the diagnostic and prognostic significance of isolated electrocardiographic abnormalities than any other agency. Current views as to the prognostic significance of bundle-branch block, for example, are based on studies carried out in hospitals, where an electrocardiographic examination is seldom made unless heart disease is obviously present or is suspected, and where facilities for following the patient after he leaves the hospital and thus learning the eventual outcome are in general unsatisfactory. Under these circumstances it is not surprising that bundle-branch block has come to be regarded as a very serious cardiac disorder. We need very badly to know what the significance of bundle-branch block may be when it is discovered in a person who displays no other evidence of a diseased heart and seems to be in perfect health. Only the insurance companies have access to a large number of electrocardiograms taken on apparently normal subjects; they alone encounter electrocardiographic abnormalities as isolated phenomena; and they alone have an entirely adequate follow-up system which makes it possible to learn the eventual outcome in almost every instance.

In the second place, I suppose I may have been invited here with the idea that I could help you solve some of the electrocardiographic problems that confront you when you are trying to decide whether an applicant for insurance should be accepted or rejected. I am afraid that I cannot help you as much as I should like to; that I shall not be able to furnish you with criteria that will enable you to decide with confidence whether a borderline electrocardiogram of one of the kinds which you so frequently encounter, is definitely inside or definitely outside normal limits. I feel that the only hope of establishing such criteria lies in a careful statistical study of the electrocardiogram carried out by the insurance companies themselves.

I may point out that the purpose served by an electrocardiographic examination in insurance work and that served by an electrocardiographic examination in clinical medicine are in general different. In the former the examiner expects the electrocardiogram to give him information which will help him to decide whether the applicant should be accepted or rejected. He cannot assume that he has the full coöperation of the applicant. He must make a definite decision one way or the other, and this decision must often be based on objective data. His chief concern is prognosis, but he is interested in the average life-expectancy of a large group rather than in the life-expectancy of the individual applicant. He must give his insurance company rather than the applicant the benefit of any doubt that may arise. The clinician, on the other hand, can assume that the patient has withheld no pertinent information. His interpretation of the electrocardiogram is often influenced to a considerable degree by the symptoms, physical findings and clinical history. He is not often compelled to make a definite decision between two opposite alternatives. The treatment which he institutes and the advice which he gives on a given occasion are not necessarily final and may be varied in this or that direction to suit the attendant circumstances. His interest in prognosis relates to the probable outcome in the individual case, and when in doubt he tends to be as optimistic as possible.

Physicians in various parts of the country sometimes send me electrocardiograms for interpretation. In examining these curves I ordinarily assume that the questions at issue are of a purely clinical kind; that there is some question as to the diagnosis or as to the advice that should be given the patient. My comments on the electrocardiogram might be quite different if I knew that, as is sometimes the case, the question of whether insurance shall be issued is involved.

I have been interested in electrocardiography for approximately twenty-five years. During that period it has increased tremendously in scope and usefulness, but even now the percentage of physicians who display a desire to become familiar with its intricacies remains relatively small. When anyone began to discuss the electrocardiogram at a medical meeting of twenty years ago, the majority of

the audience promptly walked out. I know that relatively few of you are going to be much interested in what I have to say, and if the rest of you leave or fall quietly asleep, I shall not be embarrassed because I have had that experience many times before.

I want, first of all, to review some recent developments in electrocardiography. Later, I will discuss some borderline electrocardiograms taken on applicants for insurance. These were kindly furnished me by Dr. Ylvisaker of the Prudential Insurance Company. I shall say what I think about them from the insurance standpoint, but I must remind you that my experience in the insurance field is very limited indeed.

Angina pectoris—In many cases of angina pectoris both the physical and the electrocardiographic examination are entirely negative. In other cases definite electrocardiographic abnormalities are present, and very often these furnish the sole objective evidence of heart disease. The electrocardiographic abnormality may consist in inversion of the T deflections, in broad, deformed QRS deflections indicating the presence of intraventricular block, or in pronounced deviation of the mean electrical axis to the left (right axis deviation is uncommon). These changes are, of course, in no way distinctive; they are not peculiar to angina pectoris and help to establish the diagnosis only because their presence greatly increases the probability that the cause of the symptoms lies in the heart. When the chest pain is atypical, the additional evidence which they furnish may be of the greatest value.

An electrocardiogram taken during an attack of anginal pain may show changes of a different and more distinctive kind; changes which clearly indicate that the pain is associated with a pronounced disturbance of the coronary circulation. The changes to which I refer consist in upward or downward displacement of the RS-T junction and modifications of the RS-T segment or of the T wave as a whole.

It is naturally very difficult to obtain electrocardiograms during spontaneous attacks of anginal pain, but most patients with this disorder can induce an attack by exercising. Unfortunately, effort increases the heart rate and produces minor changes in the form of the electrocardiogram when the heart is normal. In judging

electrocardiograms taken during anginal attacks induced by exertion, one must, therefore, differentiate such abnormal changes as may be attributable to myocardial anoxemia from such changes as may normally occur after exertion. Unless changes of the former kind are pronounced, which is not very often the case, this differentiation may be difficult. For this reason electrocardiographic examination during an induced attack of anginal pain has not become an important diagnostic procedure.

It is probable that it has possibilities as yet unrealized. Scherf (1) has shown that the electrocardiographic changes in question, although temporary, may outlast the pain; i.e. the pain may subside within a few minutes in cases in which the electrocardiographic changes which accompany it persist for as long as a half hour. He observed also that in cases in which exertion precipitated both anginal pain and electrocardiographic changes attributable to myocardial anoxemia, it was often possible to induce the electrocardiographic changes long after the patient's condition had so greatly improved that all attacks of pain had ceased.

It may happen that precordial or other special leads will display changes of the same kind as those seen in the standard leads, but of much greater magnitude, and that some day we may be able to detect transient myocardial anoxemia, induced by exertion or otherwise, in cases of coronary sclerosis in which anginal pain has not as yet occurred. The diagnosis of "latent" angina pectoris will then become possible.

Electrocardiographic changes of the kind under consideration are illustrated in Fig. 1. Two sets of electrocardiograms are shown. The curves of the first set are the electrocardiograms of a man, aged 35 years, who complained of attacks of anginal pain which were typical in all respects. A serological test for syphilis was positive and a very, very faint aortic diastolic murmur was present. It may be assumed, therefore, that partial obstruction of the orifices of the coronary arteries, resulting from syphilitic aortitis, was the cause of the symptoms. The first electrocardiogram (A), taken while the patient was at rest shows slight deviation of the mean electrical axis to the left but is not outside normal limits. The second (B) taken immediately after exertion and during an attack

of anginal pain shows pronounced downward displacement of the RS-T junction in leads II and III and equally pronounced changes in the form of the T deflection in lead I. Minor changes in the

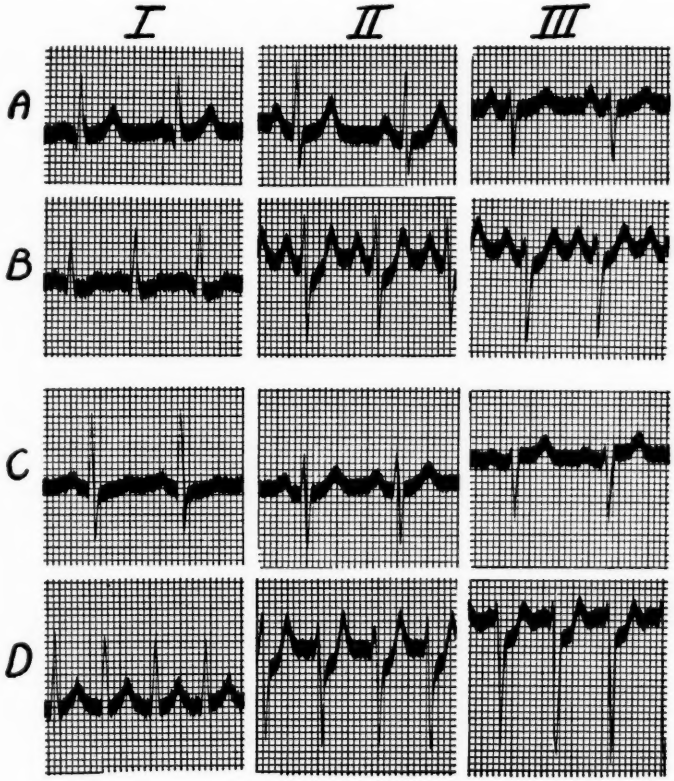


FIGURE 1. Transient displacement of the RS-T segment during an attack of anginal pain.

QRS deflections are also present, but their significance is uncertain. The patient died suddenly two or three months after these studies were made.

The last two curves of Fig. 1 are the electrocardiograms of a man, aged 53 years, who complained of chest pain which was thought to be anginal, but was not altogether typical. There was a considerable elevation of the blood pressure, but the electrocardiogram (C) was not strikingly abnormal. Since the character of the pain made me feel somewhat uncertain as to the correctness of the diagnosis, the patient was asked to walk up two flights of stairs and a second electrocardiogram was taken during the attack of pain induced in this way. This curve (D) shows very striking downward displacement of the RS-T junction in leads II and III which clearly indicates the presence of myocardial anoxemia. The heart rate is greatly increased. The P waves cannot be made out with certainty, but the form of the T waves suggests that the P and T deflections are superimposed. If this interpretation is correct the P-R interval is considerably lengthened. Note also the change in the form of the QRS deflections. As soon as these studies were completed the patient was taken to the X-ray department for a fluoroscopic examination of the chest. A few minutes later I was informed that he was dead. Death occurred without warning, and although an autopsy was not permitted, we may assume that it was due to the onset of ventricular fibrillation.

Before passing on to the consideration of another subject I should point out again that ordinarily the changes displayed by electrocardiograms taken during attacks of anginal pain are slight and difficult to evaluate. Changes of the magnitude of those shown in Fig. 1 are quite exceptional. In my personal experience pronounced changes of this kind have proved an evil omen.

Coronary Occlusion—In the last few years we have learned a great deal about the electrocardiographic changes produced by the sudden obstruction of a large coronary artery. This accident is sometimes followed by non-specific electrocardiographic abnormalities of the kinds that may be associated with heart disease of almost any type. Very often, however, it initiates a progressive modification of the ventricular complex which is highly distinctive and of the greatest diagnostic value.

Ordinarily, the first change consists in transient displacement of the RS-T junction and segment, usually more pronounced and

more lasting than that sometimes seen in angina pectoris during an attack of pain. In coronary occlusion the displacement is almost always discordant (not in the same direction in all three standard leads) whereas in angina pectoris it is frequently concordant (in the same direction in all three standard leads). Transient RS-T displacement, usually concordant, is not very uncommon in acute pericarditis.

It is imperative that transient RS-T displacement, which is apparently always due to acute injury involving a considerable fraction of the ventricular muscle, be distinguished from permanent RS-T displacement. In bundle-branch block and in axis-deviation the latter is often pronounced; in normal electrocardiograms it is not uncommon but is ordinarily slight. So far as is known, it has no important significance. The RS-T displacement produced by digitalis is a special kind; it is transient and concordant, but ordinarily involves the segment which follows the RS-T junction rather than the junction itself.

The RS-T displacement of coronary occlusion may last a few hours or a few days, rarely longer. As it subsides characteristic changes in the contour of the T deflection almost always develop. This wave undergoes a sort of invagination which begins at its end and progresses toward the RS-T junction. When the original displacement is upward the end of T begins to dip below the base line and as this process involves earlier and earlier portions of this deflection a deep V-shaped depression is produced. Where this depression joins the RS-T segment, often slightly elevated still, there is a distinct shoulder or rounded convexity directed upward. When the original displacement is downward changes of opposite sign occur and T eventually becomes a tall pointed summit. These characteristic peculiarities of the T deflections often require two or three weeks to reach their full development and usually persist for several months. They retrogress very slowly, but in many cases the T waves eventually regain a normal outline.

These changes in the final deflections of the ventricular complex are usually accompanied by equally characteristic modifications of the initial deflections. The latter begin to appear a few hours after the coronary accident and reach their height within a few

days. They may subsequently become less pronounced or disappear, but they are almost always permanent. The changes in question consist in the development of conspicuously large and often broad or notched Q deflections in those leads in which the RS-T junction is displaced upward at the onset and in which sharp inversion of the T deflection eventually appears.

As to the origin of these electrocardiographic abnormalities I may say that the RS-T displacement is due directly to electrical forces arising in the muscle supplied by the occluded artery as a consequence of the injury sustained by the semipermeable membranes surrounding its fibers. It persists until the injured muscle dies or recovers. The changes in the initial ventricular deflections are due to subtraction from the total electrical forces produced by the healthy heart, of those electrical forces formerly produced by muscle which has been killed or has been so seriously injured that it is no longer capable of responding to the cardiac impulse. The progressive changes in the T wave apparently depend upon disturbances in the recovery process in muscle that has been damaged but not killed.

At this point it is necessary to differentiate between electrocardiograms of the Q_1T_1 type and electrocardiograms of the Q_3T_3 type. In the first type (Fig. 2) lead I shows initial elevation of the RS-T junction (Fig. 2A) followed by sharp inversion of the T deflection. The QRS group in this lead begins with a conspicuous Q wave, but all of its deflections are small and the largest does not often exceed 5 mm. In lead III the initial displacement of the RS-T junction is downward and tall, upright, pointed T waves develop. The form of the QRS deflections in this lead is variable but Q is not present. The ventricular complexes of lead II sometimes resemble those of lead I in general outline, but they are much more often similar to those of lead III. In many instances large S waves are present in leads II and III, but S is frequently absent in all leads. In lead I the QRS deflections sometimes resemble the letter W in outline. Such evidence as is available at present indicates that electrocardiograms of the Q_1T_1 type are the result of recent infarction of the anterior and left lateral walls of the left

ventricle, which is due in most instances to sudden occlusion of the anterior descending branch of the left coronary artery.

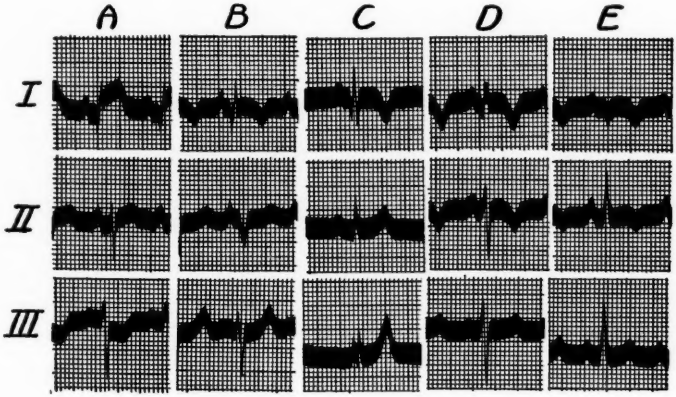


FIGURE 2. Electrocardiograms of the Q_1T_1 type, diagnostic of infarction of the anterior wall of the heart.

Infarction of the diaphragmatic wall of the heart gives rise to electrocardiograms of the Q_3T_3 type (Fig. 3). In these, leads II and III display elevation of the RS-T junction followed by the appearance of conspicuously large Q deflections and large, sharply inverted T waves. In lead I the RS-T displacement is downward and as it disappears a large upright T develops; the QRS deflections in this lead are in no way peculiar except that Q is absent or very small. In lead II the QRS deflections are often small and may look like the letter W. Electrocardiograms which show no Q deflection in lead II but are otherwise similar to curves of the Q_3T_3 class are sometimes seen. Curves of this kind may occur under circumstances which clearly indicate that they are the result of myocardial infarction, but they are much less significant from a diagnostic standpoint than curves in which a prominent Q deflection is present in lead II as well as in lead III.

It is not easy to set up criteria which will make it possible always to differentiate with certainty the electrocardiographic changes

produced by coronary occlusion which I have described from somewhat similar changes occurring in association with other types of cardiac disease. The difficulties that arise when an at-

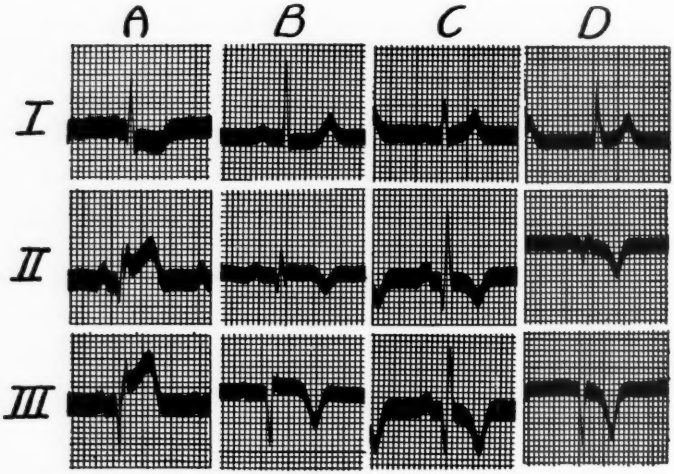


FIGURE 3. Electrocardiograms of the Q_3T_3 type, diagnostic of infarction of the posterior wall of the heart.

tempt is made to establish criteria of this kind are illustrated by a study carried out under my direction by Durant (2). Table I is taken from his paper. The first step in this study consisted in removing from the files all electrocardiograms in which lead I displayed an R wave less than 5 mm. in height and a Q wave at least 1 mm. in depth and at least one-fifth as large as the largest R wave in any of the three leads. The analysis of the case histories of the 32 patients whose electrocardiograms were of this kind, disclosed clinical evidence of coronary occlusion or of angina pectoris in about 50 per cent. of the cases. The incidence of coronary disease was raised to 73 per cent. by dropping from the series those electrocardiograms in which Q in lead I was not followed by a definite R deflection. This eliminated a number of cases of mitral stenosis in which there was conspicuous right axis-deviation, but

TABLE I—Q_1 Group.

	Criteria	Total Cases	Coronary Disease			No Evident Coronary Disease	Percentage Coronary Disease
			Coronary Occlusion	Angina Pectoris	Total		
A	Q_1 at least 1 mm. R_1 less than 5 mm. Q_1 at least 20% of largest R.	32	13	4	17	15	53.1
B	Same as under A, and in addition curves eliminated in which no R_1 is present.	23	13	4	17	6	73.9
C	Same as under B, and in addition inversion of T_1 without inversion of T_2 or T_3.	14	10	3	13	1	92.8
	Q_3 Group.						
A	Q_3 at least 25% of largest Q R S deflection. Q_3 at least 1 mm. Left axis deviation or normal axis.	96	31	29	60	36	62.5
B	Q_3 at least 50% of largest Q R S deflection. Q_3 at least 25% of R_3. Left axis deviation or normal axis.	44	25	11	36	8	81.8
C	Same as under B, and in addition T-wave inversion in leads II and III, but not in lead I.	22	18	4	22	0	100.0

no small upward movement (R) preceding the deep downward deflection of lead I. The absence of such an upward movement necessitated calling the downward movement Q instead of S, and, therefore, including these electrocardiograms in the group first selected.

The incidence of coronary disease was raised to 92.8 per cent. by eliminating all electrocardiograms which failed to show inversion of T in lead I without inversion of this deflection in leads II and III. This change in the criteria resulted, however, in the elimination of three cases of coronary occlusion and one case of angina pectoris.

There were in the files 96 electrocardiograms which displayed Q deflections in lead III which were one-fourth as large as the largest QRS deflection in any lead; Q deflections in lead II which measured at least 1 mm; and a mean electrical axis which was either rotated to the left or in the normal position. The incidence of coronary disease in the corresponding group of patients was 62.5 per cent. By eliminating all electrocardiograms in which Q in lead III was not at least one-half as large as the largest QRS deflection in any lead in which Q in lead II was not at least one-fourth as large as R in lead II the incidence of coronary disease was raised to 81.8 per cent., but 6 cases of coronary occlusion and 18 cases of angina pectoris were lost. By eliminating, in addition, all electrocardiograms which failed to show inversion of T in leads II and III without inversion of this deflection in lead I the incidence of coronary disease was raised to 100 per cent., but this change in the criteria resulted in the further loss of 7 cases of coronary occlusion and an equal number of cases of angina pectoris.

It should be pointed out that there is no reason to believe that uncomplicated angina pectoris produces electrocardiographic changes of the kind under consideration. The cases classified as angina pectoris in Table I were cases of anginal pain in which the clinical data available did not justify a diagnosis of myocardial infarction. Nevertheless, it is probable that myocardial infarction had occurred and was responsible for the electrocardiographic changes.

It will be seen that when characteristic changes in the QRS complex are combined with characteristic changes in the T wave the probability that coronary occlusion has occurred is very great. Changes in the QRS complex alone and changes in the T wave alone have less diagnostic value. Since the former are usually permanent they are, however, of the greatest importance in the diagnosis of old myocardial infarcts. In the early stages of myocardial infarction repeated electrocardiographic examination often establishes the diagnosis by disclosing a characteristic sequence of changes in the RS-T segment and T wave, when there is doubt as to the correct interpretation of the initial tracings.

Unfortunately, the electrocardiographic abnormalities described in preceding paragraphs do not begin to develop until coronary thrombosis has occurred. Immediately before this accident the electrocardiogram may be normal. The two curves shown in Fig. 4 are the electrocardiograms of a patient sent to me by a physician

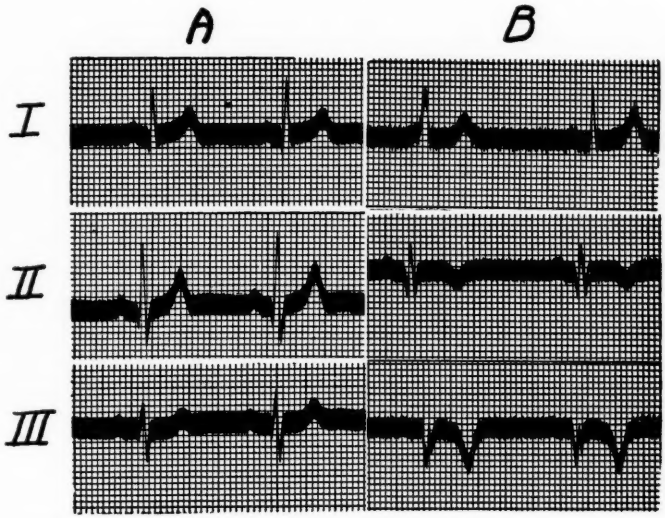


FIGURE 4. Two electrocardiograms of the same patient. The first (A) was taken a few hours before and the second (B) a few weeks after the onset of symptoms typical of coronary occlusion.

in a neighboring city. When I first saw this patient he was having severe anginal pain of recent onset, and not closely related to effort. Symptoms of this kind suggest that coronary occlusion may be imminent; they very often precede this accident. The electrocardiogram, however, was normal. Symptoms typical of coronary occlusion developed on the same day while the patient was on his way home. The second electrocardiogram, a curve of the Q_3T_3 type, was taken when he returned for a check-up examination a few weeks later. Note that the small Q deflection originally present in lead I has disappeared and that deep Q deflections have developed in leads II and III. The normal T waves of leads II and III have been replaced by deep V-shaped depressions.

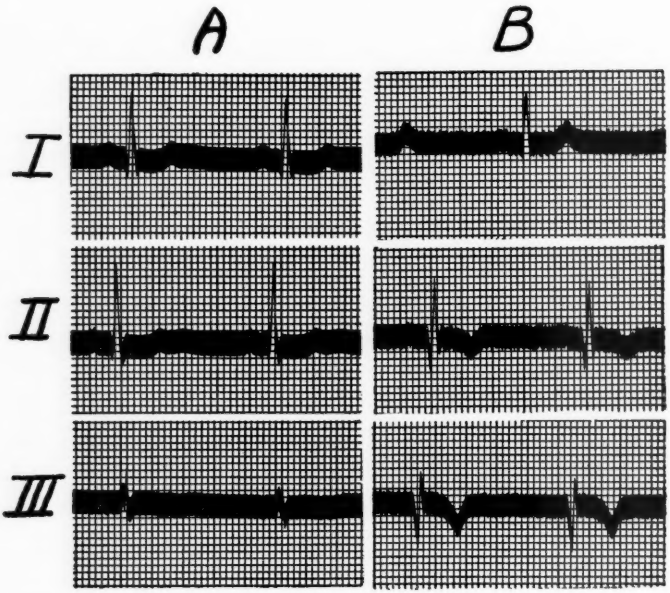


FIGURE 5. Two electrocardiograms of the same patient. In this instance electrocardiographic changes suggesting coronary occlusion developed without the occurrence of dramatic symptoms.

Similar changes in the electrocardiogram may take place without the occurrence of dramatic symptoms. The curves shown in Fig. 5 are the electrocardiograms of a man who had typical angina pectoris and also symptoms of cholecystitis. The electrocardiogram taken at the time of admission to the hospital shows flat T waves, but is not definitely abnormal. The second electrocardiogram, a curve of the Q_3T_3 type, was taken two or three weeks later after a cholecystectomy had been performed. During the intervening period no symptoms suggesting coronary thrombosis had been noted; nevertheless, the electrocardiogram clearly indicates that myocardial infarction of the diaphragmatic wall of the heart had recently taken place.

Fig. 6 illustrates an interesting sequence of changes in the electrocardiogram of a woman, who was 63 years of age when first examined on Feb. 25th, 1932. The initial electrocardiogram displays left axis-deviation with extremely large QRS deflections and inverted T waves in lead I. At this time a diagnosis of severe angina pectoris and arterial hypertension (systolic blood pressure 250 mm. Hg.; diastolic pressure, 150 mm. Hg.) was made. The second electrocardiogram (Nov. 8th, 1934) differs from the first in three particulars; the QRS deflections are slightly smaller, there is more conspicuous displacement of the RS-T junction in leads I and III, and the small R of lead III has disappeared so that the deep downward deflection of this lead must be called Q instead of S. The last electrocardiogram was taken on Nov. 17, 1934, ten hours after the onset of symptoms typical of coronary occlusion. It shows large Q waves in lead II as well as in lead III, pronounced displacement of the RS-T junction, and beginning inversion of the final portions of T in leads II and III.

The RS-T displacement shown by the second electrocardiogram and that shown by the final tracing do not have the same significance. The former is not only less pronounced, but is presumably of the permanent variety. The latter is not only extreme, but is of the transient variety, which seems to be invariably due to very recent injury to the heart muscle. The significance of the large Q deflection in lead III in the second electrocardiogram is unknown; on the other hand the presence of large Q deflections in lead II

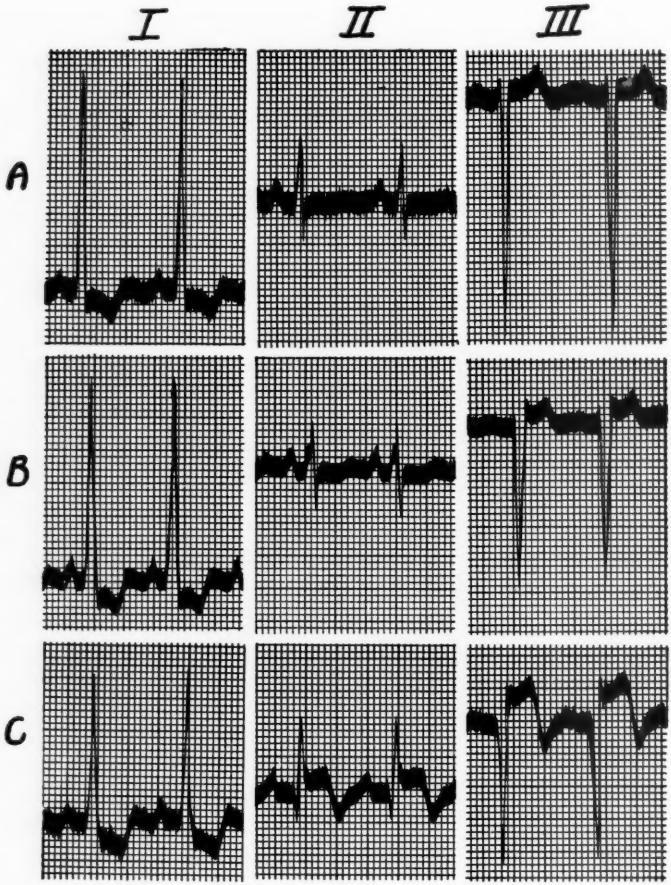


FIGURE 6. Three electrocardiograms of the same patient. The first (A) was taken Feb. 25, 1932, and the second (B) on Nov. 8, 1934. Coronary thrombosis occurred on Nov. 17, 1934, and the third tracing (C) was taken 10 hours later.

as well as in lead III in the third tracing is certainly the result of the infarction of a considerable mass of muscle in the diaphragmatic wall of the heart. It is, of course, obvious that profound electrocardiographic changes present prior to the occurrence of coronary occlusion must have an important influence upon the configuration of the electrocardiograms taken after its development. It is important to note in what respects the last electrocardiogram of Fig. 6 differs from those taken before coronary occlusion occurred and in what respects it is similar.

The importance of characteristic changes in the QRS complex in the diagonsis of old myocardial infarcts is illustrated by Fig. 7.

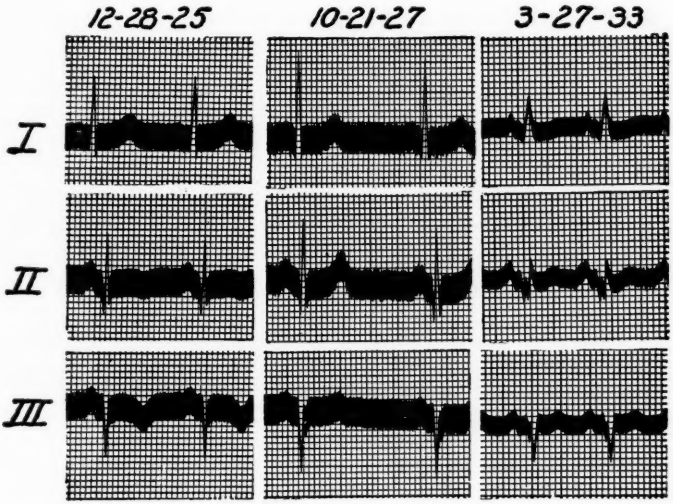


FIGURE 7. Three electrocardiograms of the same patient showing that the T-wave changes produced by myocardial infarction are often transient, the QRS changes permanent.

The first electrocardiogram was taken on Dec. 28, 1925, three weeks after the onset of symptoms typical of coronary thrombosis. It is a typical example of curves of the Q_3T_3 class. The patient was a man 59 years of age at this time. Twenty-two months later

the T waves had regained a normal outline, but the QRS complex was unchanged. Even after more than seven years the form of the initial deflections still pointed clearly to the presence of the old infarct, although the development of a slight defect in intraventricular conduction had altered some details of the QRS group.

The electrocardiographic changes produced by myocardial infarction are often of a kind much less characteristic than those I have been discussing. Non-specific electrocardiographic abnormalities, of which the most common are intraventricular block and QRS complexes of abnormally small voltage and bizarre outline, are of value in the diagnosis of myocardial infarction only when they occur in association with clinical findings which strongly suggest coronary occlusion.

The series of electrocardiograms reproduced in Fig. 8 illustrates the sudden development of small bizarre QRS deflections. The patient was a physician aged 46 years at the time of the first ex-

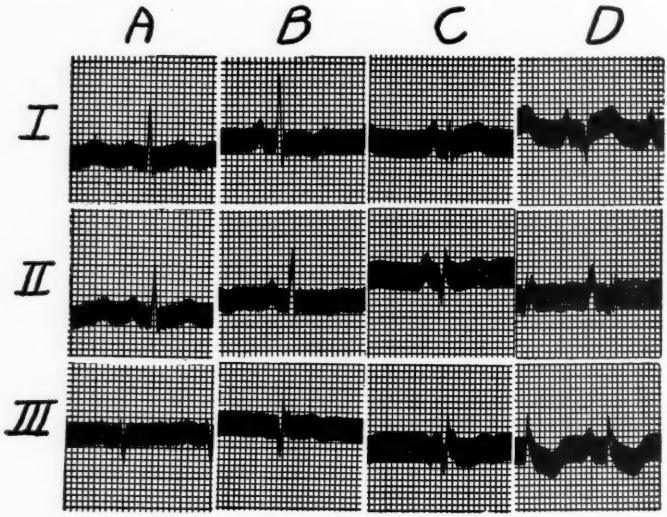


FIGURE 8. Four electrocardiograms of the same patient illustrating the sudden development of small, bizarre QRS deflections following coronary occlusion.

amination in December, 1925. At this time he had no cardiac symptoms other than palpitation due to extrasystoles, and the electrocardiogram was normal. In October, 1926, when he was having frequent but mild attacks of anginal pain on exertion, several other tracings were made. All of these show inversion of the T deflections in leads I and II and a prominent Q deflection in lead III. Coronary occlusion occurred on October 9th, 1927, while the patient was operating a lawn mower. The third electrocardiogram of Fig. 8 was taken a few hours later, and the fourth electrocardiogram of the same figure about one week after this accident. In both of these tracings the QRS deflections are extremely small and bizarre, and bear no resemblance to those of the curves taken before the onset of coronary thrombosis.

It should, I think, be emphasized that the QRS deflections in question are abnormal in shape as well as in size. The very bad reputation which low-voltage complexes have acquired rests upon studies carried out in hospitals where a large percentage of the low-voltage complexes encountered are of this kind. Electrocardiograms in which the ventricular deflections are small but normal in every other respect are not necessarily of serious import; they deserve much more careful study than they have as yet received before we can feel certain as to their significance.

Precordial Leads—In the last few years new kinds of electrocardiographic leads have come into widespread use. Precordial leads have been shown to be of great value in the diagnosis of infarction of the anterior wall of the heart and in the differentiation of right from left bundle-branch block, and of right from left ventricular hypertrophy.

Between these special leads and the standard limb leads there are certain very important differences which should be borne in mind. The standard limb leads record the time-course of the potential difference between two points, which are on opposite sides of the heart and at a great distance from it. Both points undergo potential variations of about the same magnitude, and all parts of the heart muscle contribute to these potential variations in approximately the same measure. In the case of precordial leads, on the other hand, one electrode is placed very close to the heart and the

other at a distant point. Since the electric currents generated by the heart are short-circuited by its own tissues and by the conducting medium which surrounds it, the potential variations at the surface of the heart or in its immediate neighborhood are very much larger than those which occur in the more distant parts of the body. The potential variations at the surface of the heart are roughly 50 times as large as the potential variations of the arms and legs.

In animal experiments a small electrode in contact with the heart is often paired with an electrode on one of the extremities. Under these circumstances the potential variations of the latter are entirely negligible in comparison with those of the former and contribute almost nothing to the curve recorded. Such leads are essentially unipolar; the tracings which they yield represent the time-course of the potential of the exploring electrode. The distant electrode is almost wholly indifferent and its location, so long as it is far from the heart, is of no importance. The muscle which is in contact with the exploring electrode or very close to it exerts a much greater influence upon its potential than muscle in the more distant parts of the heart and the electrical forces which it produces dominate the curve obtained. In the case of precordial leads the potential variations of the exploring electrode are much smaller than in direct leads, but they are still much larger than those of the distant electrode. Leads of this kind are semi-direct leads; they yield tracings similar to those of direct leads in general outline. These tracings are dominated by the potential variations of the exploring electrode, and hence by the activities of those portions of the heart wall with which this electrode is in close proximity.

In order that the analysis and interpretation of the electrocardiogram shall not be unnecessarily complicated, it is desirable that the two electrodes undergo potential variations of similar magnitude as in the case of the standard limb leads, or that one electrode be made as nearly indifferent as possible. In order to accomplish this purpose and thus make precordial and esophageal leads more nearly comparable to direct leads, we have devised a method of reducing the potential variations of the distant electrode to a

minimum. This method involves connecting a central terminal to electrodes on the right arm, left arm, and left leg through resistances of 5,000 or more ohms. The central terminal takes the place of the distant electrode and its potential variations are much smaller than those of an electrode placed on the back or on one of the extremities. In most cases, however, the form of the electrocardiogram is essentially the same when an esophageal or precordial electrode is paired with this central terminal as when it is paired with an electrode on the back, on the right arm, on the left arm, or on the left leg.

There has been a good deal of confusion with regard to precordial leads because different investigators have employed different methods. Some have used a large precordial electrode and others have used a small one. Some have placed the indifferent electrode on the left leg, some have placed it on the back, and some have placed it on the right arm. Some have made the galvanometer connections in one way and others in the opposite way. Recently a standard procedure has been recommended in a joint report by the Committee on Precordial Leads of the American Heart Association and the Sub-committee on Chest Leads of the Cardiac Society of Great Britain and Ireland. I need not repeat here the recommendations made in this report, which has been published in the American Heart Journal (3). I may call attention, however, to the recommendation that the galvanometer connections be made in such a way that relative positivity of the precordial electrode is represented by an upward deflection in the finished tracing. This method is the opposite of that which has been most widely used in this country. It reverses the direction of all the deflections so that the T deflections in normal precordial electrocardiograms are upright instead of inverted.

At Ann Arbor we ordinarily take a series of six precordial leads. For the first (V_1) the precordial electrode, a small copper disk about one inch in diameter, is placed in the 4th intercostal space at the right sternal margin; for the second (V_2), in the 4th intercostal space at the left sternal margin; for the third (V_3), half way between the left sternal margin and the left midclavicular line and on a line drawn from a point in the 4th space at the left sternal

margin to the apex beat; for the fourth (V_4), in the left midclavicular line at the level of the apex; for the fifth (V_5), in the left anterior axillary line at the level of the apex; and for the sixth (V_6), in the left midaxillary line at the level of the apex. The central terminal already described serves as the indifferent point. In many instances each precordial lead is taken simultaneously with standard lead I. A set of six normal precordial curves taken by this method is shown in Fig. 9. The galvanometer connections were made in such a way that positivity of the precordial electrode is represented by an upward deflection.

I may call attention, first of all, to the chief downward movement of the QRS complex in these precordial curves; it is very steep and is represented by a very thin line. This sharp downstroke corresponds to the intrinsic deflection of direct leads and its onset signals the arrival of the excitatory process at the epicardial surface of the portion of the ventricular wall nearest the precordial electrode. It will be noted that in the first two precordial leads (V_1 and V_2) this downstroke occurs early in the QRS interval, whereas in the last two it is definitely later. This difference in time is easily explained. The excitatory process reaches the ventricles by way of the His-bundle and its branches and spreads over the endocardial surface at the beginning of the QRS interval; it then spreads outward through the ventricular wall and the time of its arrival at the epicardial surface varies with the thickness of the muscle. Where the ventricular wall is thin it reaches the epicardial surface early; where the ventricular wall is thick it reaches the epicardial surface late. The epicardial surface of the thin-walled right ventricle is activated 0.02 or 0.03 second earlier than the epicardial surface of the thick-walled left ventricle. In direct leads from the right ventricle and in precordial leads from the right side of the precordium the chief downstroke or intrinsic deflection begins 0.02 or 0.03 second earlier than in direct leads from the left ventricle or in precordial leads from the left side of the precordium. When precordial leads are taken simultaneously with standard lead I, as in Fig. 9, the time of the onset of the intrinsic deflection with reference to the first deflection of lead I may be easily measured.

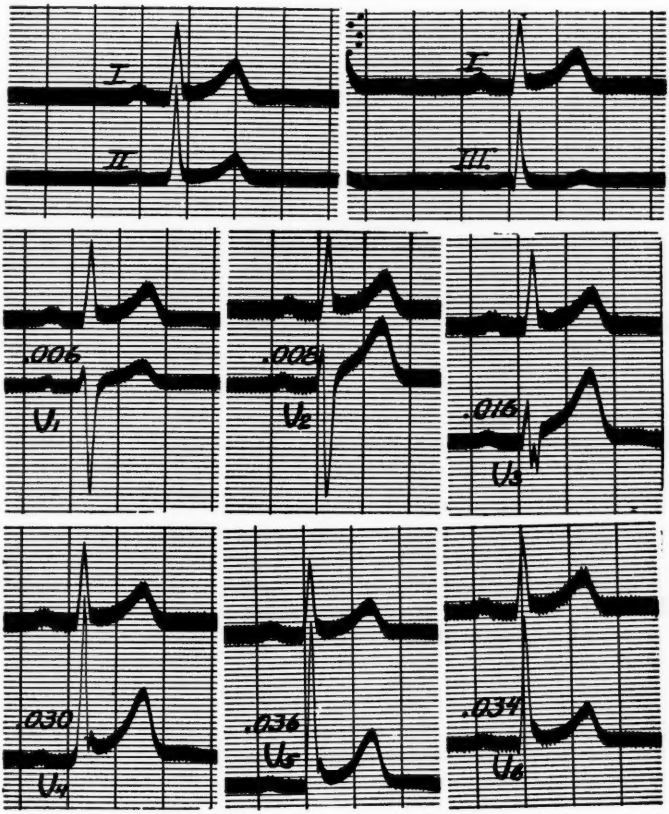


FIGURE 9. Normal precordial electrocardiograms. Lead I (above) was taken simultaneously with each precordial lead. The decimals give the time of onset of the chief downstroke or intrinsic deflection with reference to the first deflection of lead I.

The intrinsic deflection is always preceded by an upright deflection (R) which we ascribe to the electrical forces generated by the impulse in its spread outward through that portion of the ventricular wall nearest the precordial electrode. This preintrinsic

plus deflection is usually small in those leads in which the intrinsic deflection is early and large in those leads in which the intrinsic deflection is late; it is, therefore, small in the first two precordial leads and large in the last two. In leads from the left side of the precordium the preintrinsic plus deflection may be preceded by a small minus or downward deflection (Q). In normal precordial electrocardiograms this deflection is never large (not more than 3 mm. in depth), and it never occurs in the first two precordial leads (4). The intrinsic downstroke may or may not cross the baseline. When it crosses the baseline the QRS complex ends in a postintrinsic minus deflection or S wave. This wave is normally large in leads from the right side of the precordium and small or absent in leads from the left side. The QRS complex of the third precordial lead is often transitional in form between that of the leads from points farther to the right and that of leads from points farther to the left. The T-deflection is sometimes inverted in the first precordial lead, but in the electrocardiograms of normal adults it is invariably upright in the others. It has been reported that children may show inversion of the T-deflection in leads from the region of the apex beat.

We began to use precordial leads in 1929. At that time we were attempting to show that the common type of bundle-branch block is left and not right bundle-branch block, as was then commonly thought. Theoretical considerations and experiments on animals led us to believe that precordial leads would prove useful for this purpose. The results obtained in the two types of bundle-branch block are illustrated in Fig. 10. In the common type of bundle-branch block (Fig. 10 B) the onset of the chief downstroke or intrinsic deflection is early in leads from the right side of the precordium and very late in leads from the left side. In the former it occurs at about the same time, with reference to the first QRS deflection of lead I, as in normal subjects; in the latter it occurs not 0.02 to 0.03 second later as it does normally but from 0.08 to 0.10 second later. It may be concluded, therefore, that the impulse reaches the epicardial surface of the right ventricle at about the normal time, but that it reaches the epicardial surface of the left ventricle much later than is normal. Consequently, the common

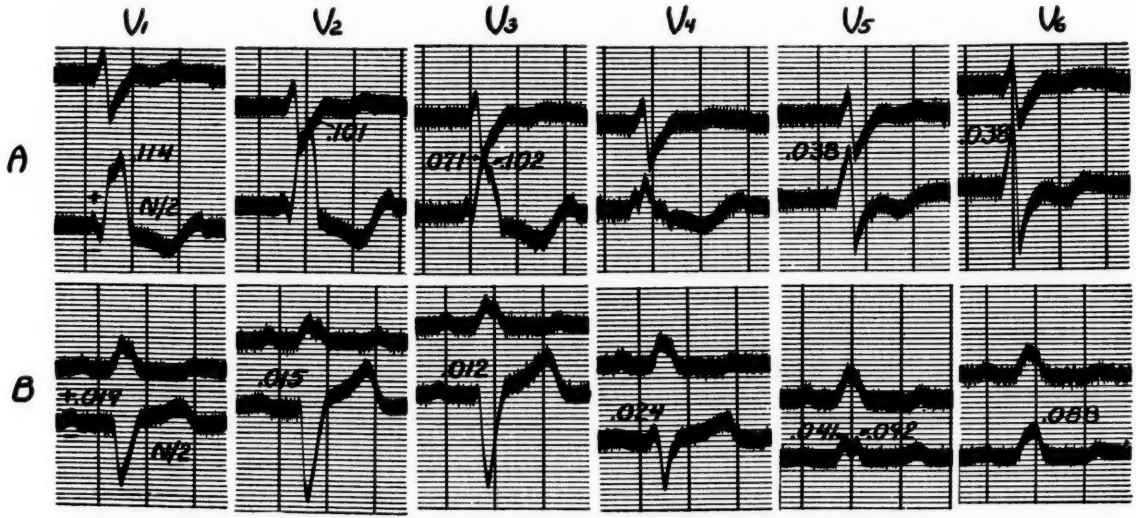


FIGURE 10. A. Right bundle-branch block. Multiple precordial leads each taken simultaneously with lead I (above). The intrinsic deflection is abnormally late in Leads V_1, V_2, and V_3.

B. Left bundle-branch block. Multiple precordial leads each taken simultaneously with lead I (above). The intrinsic deflection is abnormally late in leads V_5 and V_6.

In this and in the following figures the Symbol N/2 indicates that the precordial leads were taken with the galvanometer at one-half the usual sensitivity.

type of branch block must be left branch block rather than right branch block.

In the rare type of bundle-branch block (Fig. 10 A), on the other hand, the intrinsic deflection begins at about the normal time

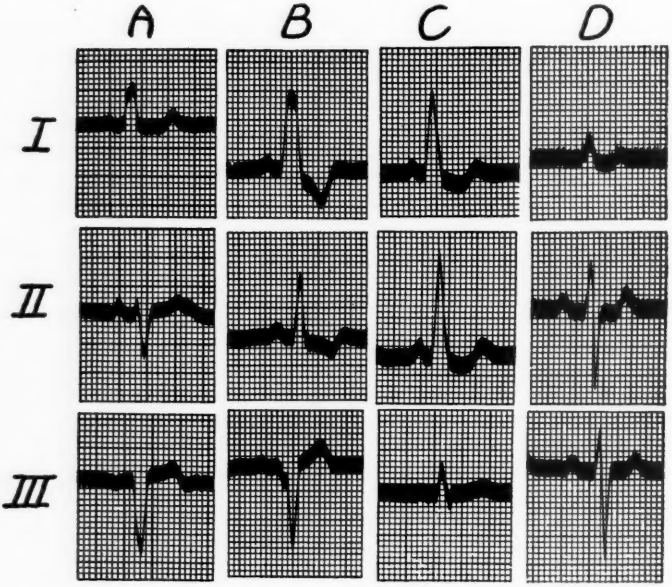


FIGURE 11. Electrocardiograms which indicate the presence of left bundle-branch block.

in leads from the left side of the precordium, but is very late in leads from the right side of the precordium. In other words the excitation wave reaches the epicardial surface of the left ventricle at the normal time and reaches the epicardial surface of the right ventricle several hundredths of a second later. This type of branch block is therefore right branch block.

This method of studying bundle-branch block has yielded very interesting results. It has been found that in practically all cases

in which the QRS interval measures 0.12 second or more and in which there is a conspicuous S deflection in lead I, the precordial electrocardiograms are of the same type and indicate the presence of right bundle-branch block. When the QRS interval measures 0.12 second or more and there is no conspicuous S-wave in lead I, the precordial electrocardiograms are of the opposite type and indicate the presence of left bundle-branch block. All of the electrocardiograms shown in Fig. 11 depict left bundle-branch block and all of those shown in Fig. 12, right branch block. It will be noted that in human branch block, both right and left, but particularly the former, the ventricular complex is extremely variable in form. In both kinds it displays a long QRS interval and conspicuous

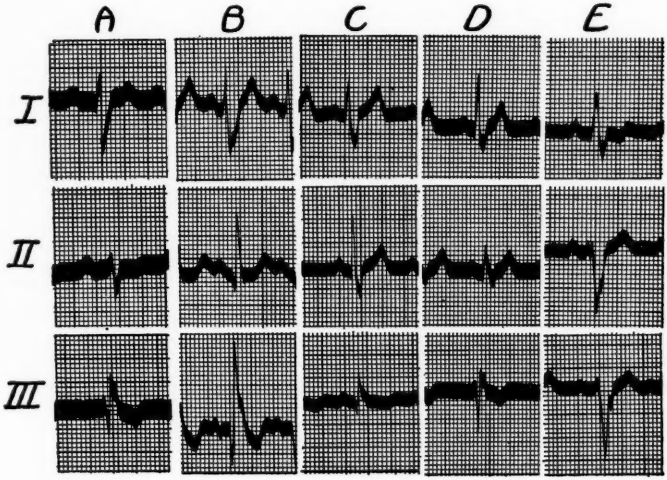


FIGURE 12. Electrocardiograms which indicate the presence of right bundle-branch block.

notching or slurring of the QRS deflections. The only feature which distinguishes between them is the conspicuous S in lead I in the curves of right branch block and its absence in the curves of left branch block.

We have used multiple precordial leads quite extensively in the study of preponderant hypertrophy of the right and of the left ventricle. Apart from the length of the QRS interval, there is a good deal of similarity between the precordial electrocardiograms obtained in cases of pronounced right ventricular hypertrophy and those obtained in cases of right bundle-branch block, on the one hand, and between the precordial electrocardiograms obtained in cases of pronounced left ventricular hypertrophy and those obtained in cases of left bundle-branch block, on the other. In right ventricular hypertrophy (Fig. 13 A) the chief downstroke or intrinsic deflection is considerably later in leads from the right side of the precordium, particularly in the first precordial lead, than it is normally and it is often later than in leads from the left side of the the precordium. The preintrinsic plus deflection R is unusually large in the leads taken farthest to the right. In left ventricular hypertrophy (Fig. 13 B) on the other hand the intrinsic deflection occurs at about the normal time in leads from the right side of the precordium, and is abnormally late in leads from the left side. These observations tend to confirm the view that an increase in the thickness of the portion of the ventricular wall nearest the precordial electrode tends to delay the onset of the intrinsic deflection, because of the increased time consumed by the excitatory process in spreading from the endocardial to the epicardial surface. In cases of great cardiac enlargement associated with a lesion of the aortic valve or with arterial hypertension the precordial curves may indicate the presence of preponderant hypertrophy of the left ventricle, when the standard leads show either no axis-deviation or a shift of the electric axis to the right. It is not possible, therefore, to predict the character of the precordial curves by examination of the standard electrocardiogram. As a general rule, however, the precordial curves show evidence of increased thickness of the right ventricular wall when the standard limb leads show pronounced right axis-deviation, and evidence of increased thickness of the left ventricular wall when these leads show pronounced left axis-deviation. In some cases of left ventricular enlargement the preintrinsic plus deflection or R wave is absent in the first two precordial leads, but the cause of this abnormality, which also

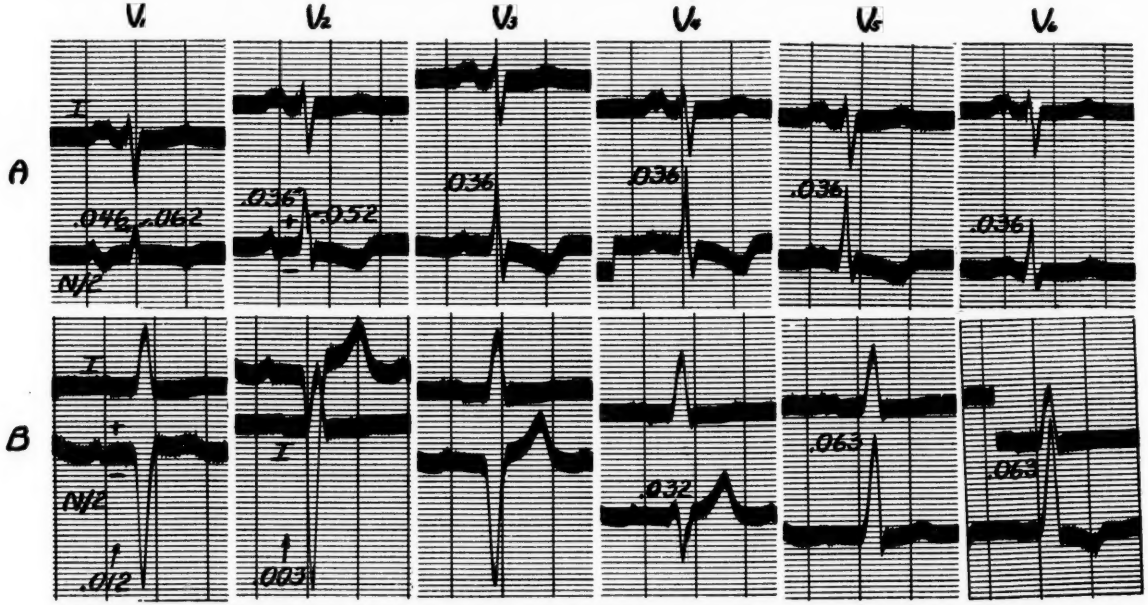


FIGURE 13. A. Right ventricular hypertrophy. Multiple precordial leads each taken simultaneously with lead I (above). The intrinsic deflection is abnormally late in leads V_1 and V_2.

B. Left ventricular hypertrophy. Multiple precordial leads taken simultaneously with lead I (above except in Lead V_3 where it is below). The intrinsic deflection is abnormally late in leads V_5 and V_6.

occurs in some cases of left bundle-branch block, is not known.

Although precordial leads are of value for many purposes, the enthusiasm which they have aroused and the popularity which they enjoy at present depends almost entirely upon their usefulness in the diagnosis of infarction of the anterior wall of the heart. They are particularly valuable in those cases in which the infarct, because of its size or location, fails to produce characteristic changes in the ventricular complexes of the standard limb leads. In infarction of the posterior wall of the heart precordial leads are of comparatively little value, and often yield entirely negative results.

The changes in the ventricular complexes of the precordial electrocardiogram produced by myocardial infarction of the ventral wall of the heart are in a general way similar to those seen in the standard limb leads. Immediately after the coronary accident there is transient upward displacement of the RS-T junction and segment (Fig. 14 A and B), which is often very pronounced. When this RS-T displacement begins to disappear the end of T becomes sharply inverted (Fig. 14 B). The inversion of T progresses toward the RS-T junction until a high V-shaped depression is produced (Fig. 14 C and D). These changes in the final deflection of the ventricular complex are usually associated with changes in the QRS group which begin at about the same time. In many instances the normally diphasic or triphasic QRS complex is converted into a single deep downward deflection or Q-wave (Fig. 14 A, B & C), sometimes notched at its onset. This transformation involves the complete disappearance of both the preintrinsic plus deflection (R) and the intrinsic deflection itself. It is ascribed to complete elimination of the electrical forces produced by the outward spread of the cardiac impulse through the portion of the ventricular wall nearest the precordial electrode. It indicates that the infarct extends from the endocardial to the epicardial surface and that the infarcted muscle is either dead or so badly damaged that it cannot transmit the excitation process. In other instances the preintrinsic plus deflection (R) and the intrinsic deflection are not abolished, but merely greatly reduced in size. In this case the QRS complex always begins with an ab-

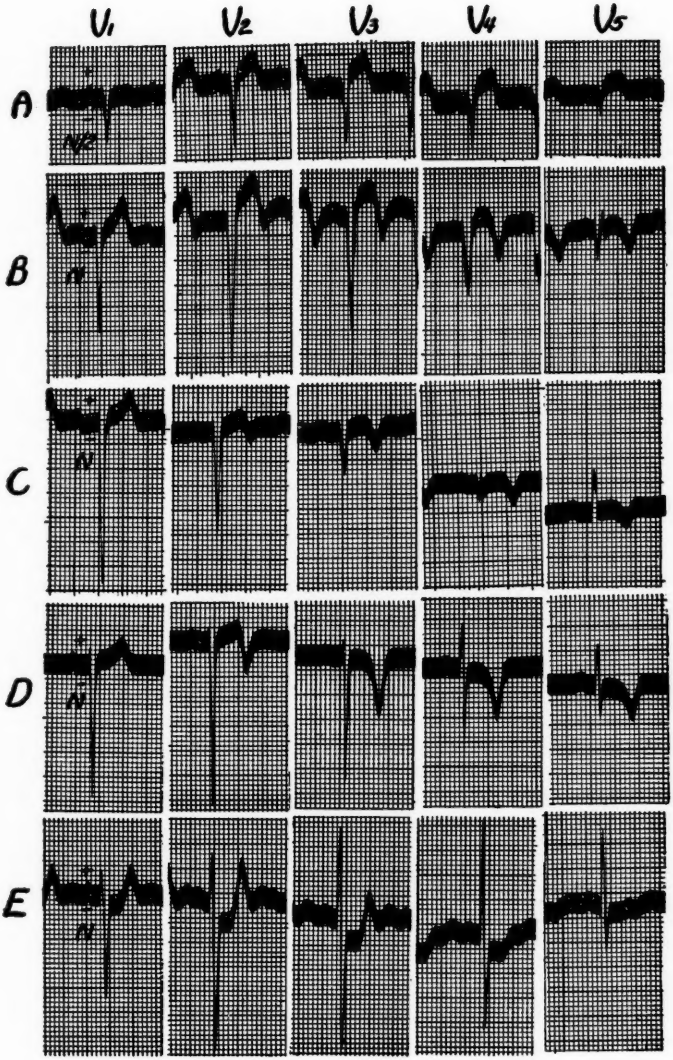


FIGURE 14. A, B, C, D. Precordial electrocardiograms diagnostic of infarction of the anterior wall of the heart.

E. Precordial electrocardiogram obtained a few hours after the onset of myocardial infarction of the posterior wall of the heart.

normally deep Q-wave and usually resembles the letter W in outline (Fig. 14-C-V_4). Complexes of this kind appear to be associated with infarcts that involve the inner layers of the ventricular wall, but do not extend to the epicardial surface.

When multiple precordial leads are taken these changes may be present in all of them or in one or two only. Displacement of the RS-T junction and inversion of T are often present in leads which fail to show characteristic changes in QRS and they are particularly likely to extend to leads taken farther to the left than those in which the QRS changes are best developed.

I have already mentioned that in some cases of left ventricular enlargement and in some cases of left bundle-branch block the preintrinsic plus deflection (R) is absent in the first two precordial leads. In these instances the QRS complex of these leads consist of a single deep downward deflection or Q wave. The presence of QRS complexes of this kind in the first two precordial leads only is not, therefore, diagnostic of myocardial infarction. Abnormally large Q waves in the leads taken farther to the left are very seldom seen in any other condition, but Master (5) has recently pointed out that they may be present temporarily in acute nephritis with cardiac damage. Very small R deflections, not more than 1 or 2 mm. in height, in one or more of the last four precordial leads (Fig. 14 D), suggest the presence of an anterior infarct, but are much less significant than the presence of an abnormally large Q deflection. The same may be said of small QRS complexes of bizarre outline in these leads.

When both the initial and the final deflections of the ventricular complex are altered in the characteristic manner a diagnosis of coronary occlusion can be made with reasonable certainty from the electrocardiogram alone. Some caution must be used in the interpretation of precordial electrocardiograms which display characteristic changes in the initial deflections only, or in the final deflections only, particularly when there are no clinical data pointing to coronary occlusion.

In the earliest stages of infarction of the posterior or diaphragmatic wall of the heart precordial leads often show conspicuous downward displacement of the RS-T junction and segment (Fig. 14 E).

After the disappearance of RS-T displacement they are usually negative, so far as distinctive signs of myocardial infarction are concerned. It should, however, be pointed out that in some cases in which the standard electrocardiogram is of the Q_3T_3 type, precordial leads show clear evidence of infarction of the anterior wall of the heart. It is possible that in such cases both surfaces of the heart are involved.

Esophageal Leads—If an electrode, placed in that part of the esophagus which lies very close to the posterior wall of the left auricle, is paired with a central terminal or with another electrode attached to one of the extremities very large auricular waves are obtained (6). A record obtained in this way from a patient with partial heart block is reproduced in Fig. 15. It will be noted that the P wave is taller than the largest QRS deflection. The extremely sharp downward movement which begins at the apex of this wave

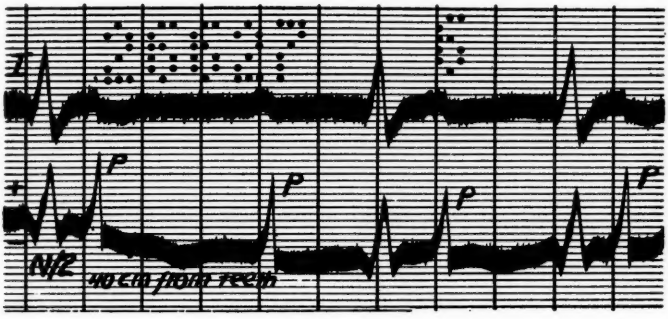


FIGURE 15. Partial heart block. Esophageal lead (below) taken simultaneously with lead I. The esophageal electrode was close to the wall of the left auricle.

is the intrinsic deflection. It marks the passage of the impulse through the part of the auricular wall nearest the esophageal electrode.

When the electrode in the esophagus is placed at a lower level and is only a short distance above the diaphragm a semi-direct lead from the base of the left ventricle is obtained. Leads of this kind

may prove to be useful in the detection of old myocardial infarcts located on the posterior wall of the heart. The esophageal electrocardiogram shown in Fig. 16 shows that they are sometimes helpful in other ways. The upper curve of this figure, which is standard

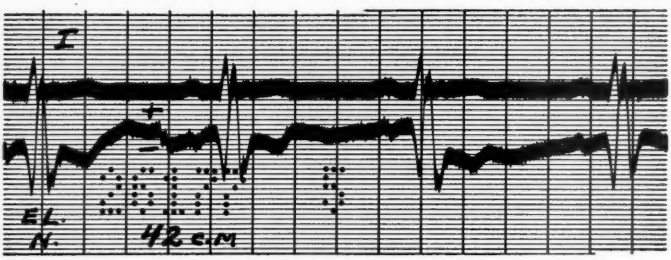


FIGURE 16. Esophageal lead (below) taken simultaneously with lead I. The esophageal electrode was close to the wall of the left ventricle. Note the broad QRS deflections of the esophageal lead, which indicate that the small summit which follows the chief deflections of lead I is really a part of the QRS group.

lead I, shows QRS deflections which appear to be of normal duration. On careful examination, however, it will be noted that the isoelectric segment which follows the main QRS deflections is interrupted by a tiny peak. The other standard leads, and precordial leads as well, show ventricular deflections of similar character, QRS deflections of apparently normal duration followed by a small deflection of unknown origin. The esophageal lead, on the other hand, shows a very broad QRS complex, and clearly demonstrates that the tiny, troublesome deflection seen in lead I is really a part of the QRS group. Intraventricular block was evidently present and some portion of the posterior heart wall was activated extremely late.

Borderline Electrocardiograms—The insurance examiner encounters many electrocardiograms which are neither definitely normal nor definitely abnormal, and, like everyone else, finds the evaluation of such curves extremely difficult. The problem which they present can not easily be solved by those who, like myself,

are operating the electrocardiographic laboratory of a hospital. Consider, for example, the lesser grades of left axis-deviation. If I should remove from my files all the tracings which show this peculiarity, and also examine the corresponding case histories, I might well find that the great majority of the patients from whom these tracings were obtained were obese and had arterial hypertension. I should, in that case, be left with the impression that most, if not all, of the electrocardiograms in question were significantly abnormal. The association between slight left axis-deviation, obesity, and hypertension might, however, be due to the selection of patients for electrocardiographic examination. Few patients are referred to my laboratory unless it is suspected that they have heart disease, and a very large percentage of those who are referred are obese middle-aged persons with an elevated blood pressure. It is in cases of this kind that the internes are most likely to feel uncertain as to the presence of cardiac involvement, and to request an electrocardiogram. The insurance examiner may frequently encounter similar grades of left axis-deviation in the electrocardiograms of applicants for insurance who appear to be in perfect health.

I believe that if the insurance companies are going to make intelligent use of the electrocardiogram in the examination of applicants for insurance they must themselves determine the significance of the various types of borderline electrocardiograms. The final solution to this problem will, of course, not be possible until it is known whether the mortality from heart disease is or is not greater for those who show the electrocardiographic peculiarities in question than for the general population.

Many years must elapse before mortality statistics of the kind needed become available. In the meantime, I believe that the insurance companies would do well to undertake a comprehensive statistical study of the normal electrocardiogram. Such a study would make it possible to determine accurately the expected frequency in normal electrocardiograms of a P-R or QRS interval of a given length, or of a P, Q, R, S, or T deflection of a given size in a given lead, and thus to estimate the probability that an electrocardiogram under consideration was abnormal. It would

then be possible to treat borderline electrocardiograms systematically and to make consistent decisions.

To illustrate what I have in mind, I may present briefly some of the results of an unpublished statistical study carried out by Nyboer and myself. This study was based on the electrocardiograms of 104 normal men, most of whom were between 20 and 30 years of age. In analysing the data obtained by measuring these electrocardiograms we made use of the Hollrieth statistical machines available in the University laboratories. Without such machines statistical studies, if at all extensive, are extremely laborious. The statistical constants determined were (a) the mean, (b) the standard deviation, and (c) the skewness. These are the constants needed in order to make use of the "Tables for Statisticians" prepared under the direction of Professor Carver and published by Edwards Brothers, Inc. of Ann Arbor.

Table II gives these three statistical constants for each of the frequency distributions obtained by measuring (a) the voltage of the P, Q, R, S, and T deflections in each of the standard limb leads; (b) the voltage of R plus S in each of the standard leads; and (c) the axis-deviation index, obtained by subtracting the sum of R_3 and S_1 from the sum of R_1 and S_3. The largest and smallest observed values of each of these variables are also given. The last four columns of the table give the values of each variate which, it is predicted, will not be exceeded more often than once in a hundred, once in a thousand, once in ten thousand, and once in a hundred thousand observations, respectively. These predictions, of course, refer to curves of the kind upon which the study was based, the electrocardiograms of normal men in the third decade of life.

Certain features of Table II require further explanation. The statistical tables available gave no data for frequency distributions with a skewness greater than 1.1. In four instances the computed value of the skewness exceeds this figure; in the case of Q in lead I it is 1.25; in the case of R in lead I it is 1.56; in the case of S in lead III it is 3.37; and in the case of R plus S in lead I it is 1.35. The first of these large values is apparently a consequence of the absence of Q_1 in 50 of the 104 curves of the series. The second is

TABLE II.

	Mean	Stand. Dev.	Skew.	Min.	Max.	1 percent.	0.1 percent.	0.01 percent.	0.001 percent.
P_1	0.50	0.21	0.87	0	1.2	1.12	1.42	1.70	1.97
P_2	1.15	0.40	0.56	0.3	2.5	2.25	2.73	3.17	3.58
P_3	0.77	0.39	0.23	0.2	1.8	1.81	2.09	2.39	2.66
Q_1	0.36	0.45	1.25	0	2.0	1.75	2.46	3.14	3.81
Q_2	0.58	0.59	1.02	0	2.5	2.36	3.25	4.09	4.91
Q_3	0.61	0.66	1.12	0	3.0	2.64	3.69	4.69	5.67
R_1	5.73 5.60	2.74 2.40	1.56 0.73	1.5 1.5	19.4 12.0	14.20 12.36	18.53 15.44	22.66 18.25	26.72 20.91
R_2	11.90	3.96	0.43	4.0	23.6	22.28	26.43	30.12	33.48
R_3	7.99	4.41	0.37	1.0	20.0	19.54	24.17	28.28	32.02
S_1	1.78	1.27	0.61	0	6.0	5.29	6.81	8.19	9.49
S_2	1.83 1.77	1.52 1.40	1.00 0.59	0 0	8.0 6.0	6.42 5.63	8.72 7.31	10.87 8.84	12.99 10.27
S_3	1.39 1.28	1.67 1.23	3.37 0.67	0 0	13.0 4.5	6.55 4.75	9.19 6.32	11.71 7.76	14.18 9.13
T_1	2.05	0.97	0.94	−0.5	5.5	4.92 0.44	6.31	7.61	9.17
T_2	2.99	1.37	0.30	0	8.0	6.47 0.10	7.81	8.98	10.05
T_3	1.22	1.22	0.30	−2.0	5.5	4.31 −1.34	5.51	6.55	7.50
$(R + S)_1$	7.58	2.94	1.35	2.5	20.6	16.66 3.11	21.31 2.64	25.75 2.43	30.10 2.28
$(R + S)_2$	13.88	3.63	0.58	6.0	25.4	23.90 7.05	28.25 5.63	32.21 4.70	35.91 4.04
$(R + S)_3$	9.68	4.27	0.51	3.2	18.0	21.17 1.35	25.95	30.26	34.28
Index	−2.68	6.70	0.31	−17.2	20.5	14.34 −16.75	20.90 −20.57	26.60 −23.45	31.83 −25.73

due to the occurrence in one of the electrocardiograms of an R deflection in lead I which measured 19.4 mm. The elimination of this very unusual curve reduced the maximum observed height of R to 12.0 mm. and the skewness of the distribution to 0.73. Another electrocardiogram displayed a striking degree of left axis deviation with S_2 deflections of 8 mm. and S_3 deflections of 13 mm. Although examination of the subject from whom this curve was obtained showed no clear evidence of heart disease, we suspected that this electrocardiogram was abnormal. When it was dropped from the series the largest S_3 remaining measured only 4.5 mm. and the skewness for S_3 fell to 0.67. In the case of S_2 the maximum observed amplitude fell from 8 to 6 mm. and the skewness from 1.00 to 0.59. It should be pointed out that relatively few observations will give a fairly accurate estimate of the mean; a larger number are needed to obtain an equally accurate estimate of the standard deviation, which is based upon the squares of the deviations of the individual variates from the mean; and a far larger number are required to obtain a reasonably accurate estimate of the skewness, which is based on the cubes of the deviations from the mean. It will be noted that casting out the largest variate, in the instances mentioned, had a much greater effect upon the skewness than upon the other statistical constants. In order to obtain entirely reliable values for the skewness many more observations than were available would have been required.

These considerations explain the double entries corresponding to R_1, S_2, and S_3. The first entry is based upon the complete series, the second shows the effect of dropping the largest variate. The predictions of the last four columns were computed from the data given in the statistical tables for a skewness of 1.1 in those instances in which the estimated value of the skewness exceeded this figure. In the case of the index, of the sum of the R and S deflections in lead II, and of the T deflection in leads I, II, and III double entries appear in the last four columns only; the two figures give two values of the variable, the first greater and the second less than the mean, which may be expected to be equally rare.

In electrocardiography, as in other fields, it is not possible to draw a sharp line between the normal and the abnormal. In

reaching a decision as to the abnormality of a given finding we must depend upon a conscious or unconscious estimate of the probability of its occurrence in the electrocardiogram of a normal subject. Statistical tables of the kind under consideration are certainly a far better guide than the rather vague and changeable impressions that we gain by experience. In using such a table we must, of course, decide how uncommon in normal curves a given finding must be before the probability that it is an abnormal finding becomes sufficiently great to approach certainty for practical purposes. After careful examination of Table II, it is my own impression that a finding which may be expected in less than one per cent. of normal electrocardiograms should be regarded as probably abnormal, and that one which may be expected in less than one-tenth of one per cent. of normal electrocardiograms should be regarded as almost certainly abnormal.

I feel sure, however, that we need a much more comprehensive statistical study of the normal electrocardiogram than has yet been attempted. My own experience with statistical methods is so meagre that I shall not attempt to give detailed directions for making a study of the kind that is needed. I may, however, venture to make a few suggestions.

I believe that an adequate study should be based on normal subjects of both sexes and of all ages and that it should include measurements of the heart rate, the P-R interval, the QRS interval, possibly the Q-T interval, the voltage of P, Q, R, S, and T in each of the standard limb leads and in one or more standard precordial leads, the voltage of the largest deflection of the limb leads, and the preponderance index. In addition to the statistical constants for these frequency distributions it would probably be desirable to determine a number of correlation coefficients; the correlation between the age and the preponderance index, the correlation between this index and the deviation of the observed from the ideal body-weight, the correlation between the P-R interval and heart rate and between the QRS interval and heart rate, and probably others. In the study carried out by Nyboer and myself it was found that there was a definite positive correlation between the voltage of R and the voltage of Q, and a definite negative correlation between

the voltage of R and the voltage of S in all of the standard limb leads and in precordial leads as well. This indicates that large Q waves tend to be associated with large R waves, and large S waves with small R waves.

Slight Prolongation of the P-R Interval—Einthoven introduced the practice of taking electrocardiograms at a film speed of 25 mm. per second and most laboratories still adhere to it. For technically perfect curves this speed is perhaps sufficiently fast, but for the accurate measurement of the P-R and QRS intervals of the average curve a speed of 50 mm. per second is far superior.

The behavior of the P-R interval in atrioventricular rhythm, in which it may be zero or may be replaced by an R-P interval, indicates that the time required by the cardiac impulse to pass from auricles to ventricles is controlled to a very large extent by the conductivity of the atrioventricular node. This node is well supplied with nerve fibers and both vagal and accelerator tone influence the rate at which it transmits the excitatory process. The conductivity of the node seems, moreover, to be quite easily modified by other factors.

There is a very definite negative correlation between the P-R interval and the heart rate so that when the heart rate is slow the P-R interval is relatively long. This interval is easily increased in normal subjects by the administration of digitalis, which seems to produce this effect chiefly through its influence upon vagal tone, but partly by a direct effect upon the nodal muscle.

The P-R interval is very often temporarily prolonged in rheumatic fever, less often in other infections. Permanent prolongation may be seen in almost any type of heart disease; in congenital heart disease, in association with valve lesions of rheumatic origin, in syphilitic heart disease, and very often in arteriosclerotic heart disease. Occurring under these circumstances an increased P-R interval is no doubt rightly considered evidence of myocardial involvement.

The significance of a prolonged P-R interval which occurs as an isolated sign, unaccompanied by other evidence of cardiac disease, is not known. It is possible that it is frequently the result of a previous attack of rheumatic fever, diphtheria, typhoid fever,

trichiniasis, or some other infection of a kind often accompanied by myocardial involvement, but there is very little evidence which supports this hypothesis. Whether those who display this sign are more likely than others to die of heart disease is also uncertain.

The P-R interval of normal adult subjects sometimes measures no more than 0.12 second; ordinarily it is about 0.16 second, slightly more or less, unless the heart rate is very slow when it is likely to be longer. It seldom reaches 0.20 second, which is ordinarily considered the upper limit of normal. No statistical study is available which gives an adequate idea of the frequency of P-R intervals of 0.21 and 0.22 seconds in the electrocardiograms of persons who appear to be in good health, but I believe that they are not extremely uncommon and I doubt that they have any serious prognostic significance. I am, however, under the impression that P-R intervals of 0.24 second or more are rarely seen in the absence of heart disease, and that the probability that they are always abnormal is great.

Two electrocardiograms showing slight prolongation of the P-R interval are shown in Fig. 17. The first is the electrocardiogram of a man, aged 47 years, who gave a history of painful joints with fever at the age of 35. On examination of the heart there was a soft, inconstant, apical systolic murmur, which became quite loud and harsh after exertion. The P-R interval measures approximately 0.22 second. In this instance, there is a suspicion that this long interval is the result of a previous attack of rheumatic fever. The second tracing is the electrocardiogram of a man, aged 60 years, who was apparently in good health. Here, the P-R interval measures nearly 0.24 second. In this instance the possibility that the long interval might be the result of early arteriosclerotic changes would have to be given serious consideration.

Slight Prolongation of the QRS Interval—Prolongation of the QRS interval is evidence of a conduction defect beyond the bifurcation of the His-bundle; either in one of the two main branches of the bundle, or in the subdivisions of these branches. Intraventricular conduction is not influenced by vagal tone, or by digitalis, but may be depressed by quinidin. There is a negative correlation between the QRS interval and the heart rate, but the magnitude of

the correlation coefficient has not been determined. On the whole a prolonged QRS interval is a considerably more reliable sign of cardiac disease than a prolonged P-R interval. There is a peculiar condition in which a long QRS interval is associated with a very

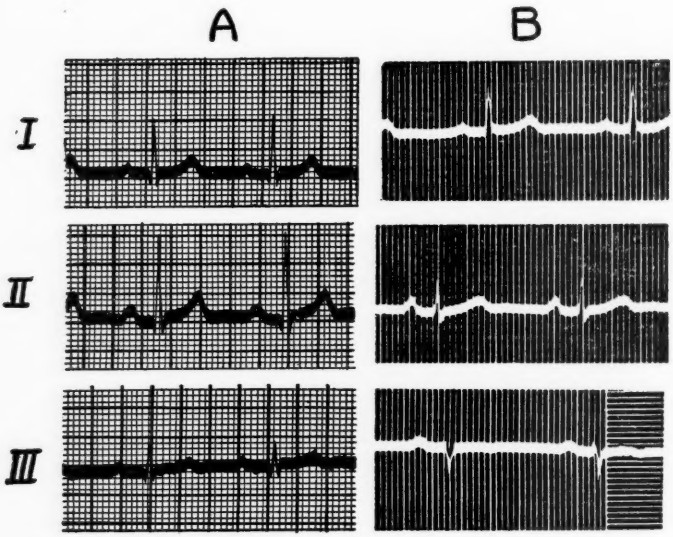


FIGURE 17. Two electrocardiograms which show slight prolongation of the P-R interval.

short P-R interval. In this condition the administration of atropin, or the reduction of vagal tone in some other way may result in a return of these intervals to their normal values. Those who display electrocardiograms of this kind are subject, as a rule, to attacks of paroxysmal tachycardia, but often show no other evidence of heart disease. I do not consider this combination trivial. One of my patients who displayed these phenomena died during a prolonged attack of tachycardia, and definite myocardial changes were found at autopsy.

The normal QRS interval ordinarily measures 0.06 to 0.08 second. Intervals as long as 0.10 second, which is regarded as the upper limit of normal, are decidedly uncommon in the electrocardiograms of young subjects who are in good health. When the QRS interval is definitely longer than 0.10 second the probability that one is dealing with an abnormal tracing is so great as to approach certainty. It should, however, be pointed out that it is frequently difficult to estimate the QRS interval accurately, because of uncertainty as to the exact point at which the QRS deflections begin or end, and also that when the QRS complex is abnormally long it is seldom normal in contour. It is almost always notched or slurred or shows other peculiarities in form.

The first tracing of Fig. 18 is the electrocardiogram of a man, aged 51 years, who gave a long and complicated history of gastrointestinal disturbances, but showed no evidence of cardiac disease.

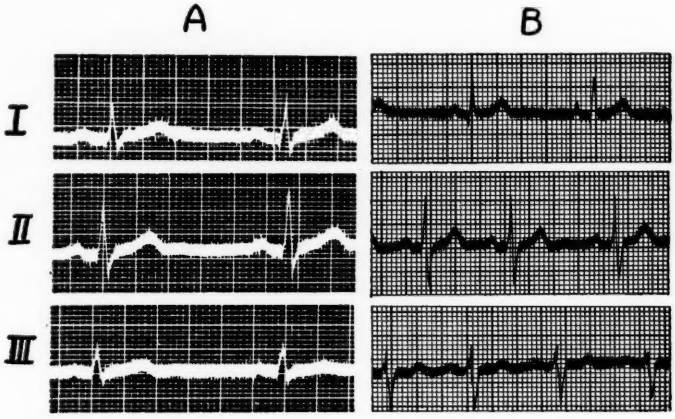


FIGURE 18. Two electrocardiograms which show questionable prolongation of the QRS interval.

In this instance the general contour of the ventricular complex is normal. The duration of the QRS group appears to be at least 0.10 second and it may be a little longer. Both the beginning and

the end of this group are ill defined and it is impossible to measure the QRS interval satisfactorily. I would hesitate to call this an abnormal curve, but would advocate another electrocardiographic examination with a film speed of 50 mm. per second.

The second tracing of Fig. 18 is the electrocardiogram of a man, aged 31 years, who appeared to be in perfect health. It shows some peculiarities of form; there is slight left axis deviation, but both the index and the voltage of S_3 are well within normal limits. The splintering of the first part of QRS in lead III is not abnormal. The QRS complex does not appear to be unusually wide in leads I and II, but in lead III its duration is certainly 0.10, and perhaps 0.11 second. Here again, I should advise another electrocardiographic examination with a faster film speed. I think it unlikely that this electrocardiogram is abnormal.

Low Voltage QRS Deflections—The voltage of the QRS complex is ordinarily considered abnormally small when the excursion of its largest deflection measures less than 5 mm. in all of the standard limb leads. In the clinic QRS deflections of this kind are most often associated with recent coronary occlusion, high grade cardiac failure with edema, arteriosclerotic heart disease, or myxedema. In the vast majority of those instances in which serious cardiac disease is present the QRS deflections are not only small but bizarre in outline; very often the QRS interval is prolonged indicating that intraventricular block is present. In myxedema small QRS deflections of normal outline occur, but they are very frequently combined with inverted T-waves, and with a noticeable lack of the small rapid oscillations due to tension of the voluntary muscles.

It is doubtful whether electrocardiograms which appear to be normal apart from the small size of the QRS deflections have an important significance unless the reduction in voltage is extreme. I believe that it is important to consider not only the magnitude of the largest displacement of the trace from the isoelectric level, but also its total excursion, or, in other words, the sum of the largest upward and the largest downward movement of any lead. The electrocardiograms of some apparently normal subjects display QRS complexes in which both R and S measure less than

5 mm. but are nearly equal in size. Table II indicates that the sum of R and S in lead II may be expected to fall below 7 mm. in about one per cent. of the curves obtained from normal young men. In my opinion, little significance should be attached to electrocardiograms of normal outline in which the largest individual QRS deflection measures slightly less than 5 mm., particularly when the total excursion of the QRS group is within normal limits.

The first curve of Fig. 19 is the electrocardiogram of a man aged 53 years who showed no clinical evidence of heart disease. The ventricular complex is of normal outline, and the sole unusual

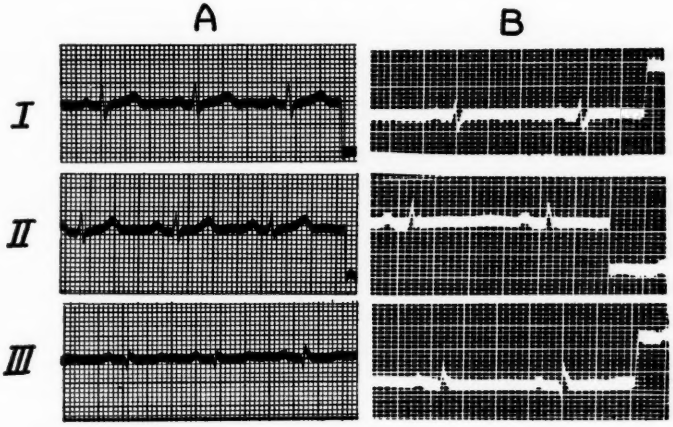


FIGURE 19. Electrocardiograms showing low voltage deflections.

feature is the small size of the QRS deflections. These are largest in lead I in which R measures 3.5 mm. and S about 2.3 mm. The total excursion in this lead is, therefore, 5.8 mm. In lead II, R measures about 3 mm. and S about 1.8 mm., so that the total excursion is 4.8 mm., a value which according to Table II may be expected to occur in about one normal electrocardiogram in 10,000. One difficulty here is that we have no data which would enable us to predict how rarely one may expect to encounter a normal electrocardiogram in which the largest R deflection in any limb lead does

not exceed 3.5 mm. and in which the largest total excursion does not exceed 5.8 mm. It should be mentioned that the deflections of the single precordial lead which was taken were not unusually small, and it is quite likely that no cardiac abnormality was present in this case. Nevertheless, until we have more information than is now available an electrocardiogram as unusual as this one, if it be considered normal, must be regarded with suspicion, particularly when the subject is past middle age.

The second tracing of Fig. 19 is the electrocardiogram of a young woman, aged 21, who had always been well and had been active in athletics. Physical and X-ray examinations of the heart were entirely negative. A number of curves were taken and in all of them the QRS deflections are small and the T-waves practically isoelectric. In a curve taken while the breath was held after forced expiration the T waves in lead I became definitely inverted toward the end of the experiment. In the single chest lead taken the largest QRS deflection measures 14 mm. and the T waves are normal. Here again the small size of the ventricular deflections is the sole unusual feature; the QRS deflections are not peculiar in contour. In all probability this is a very unusual normal electrocardiogram. The total excursion, R plus S, in lead II does not exceed 3 mm., a value which, if we may depend upon Table II, one may expect to encounter no more than once in a hundred thousand observations on normal subjects.

In regard to the problem presented by this electrocardiogram it may be said that in the first three decades of life, electrocardiographic abnormalties are not often encountered in the absence of other evidence of cardiac disease, except during the course of an acute illness. After the fourth decade heart disease without physical or X-ray evidence of cardiac abnormality is very much more common, and the probability that some unusual feature of the electrocardiogram is due to the presence of myocardial disease is very much greater.

Small T-waves—Regarding electrocardiograms in which the height of the T-waves is less than 1 mm. in all of the standard limb leads, little need be said. If one may judge from the statistical constants given in Table II, one may expect to find T-deflections

of this size in lead I in about 12 per cent., and in lead II in about 6 per cent. of a series of normal electrocardiograms. The data given in Table II do not make it possible to predict how often T waves less than 1 mm. in height might be found in both lead I and lead II. Experience indicates, however, that nearly isoelectric T-waves in both these leads are not exceedingly uncommon in the normal electrocardiogram. In rare instances slight inversion of T in lead I or II may be found in the electrocardiogram of a young adult who appears to be in perfect health, but for practical purposes it is best to consider inversion of T in these leads always abnormal.

Axis-deviation—The electromotive force produced by the heart has both magnitude and direction, and is consequently a vectorial quantity, which may be represented by an arrow of appropriate length so drawn as to make the proper angle with the horizontal or X-axis. During the inscription of the QRS deflections this cardiac vector varies from instant to instant in both magnitude and direction, and often shifts through more than 180 degrees. When the heart is normal the angle which it makes with the horizontal at the time when its magnitude is greatest ordinarily lies between 0 and 90 degrees. Because the corresponding peaks of the three leads are seldom synchronous this angle can not be determined accurately unless two leads are taken simultaneously. If one desires to determine the approximate amount of axis-deviation by inspection of the electrocardiogram, the position of the major electrical axis, which this angle defines, is more difficult to estimate than the position of the mean electrical axis.

The mean QRS deflection in any lead is equal to the net area of the QRS deflections divided by the true QRS interval. Since this interval must be the same for all leads, the QRS area is equal to the mean QRS deflection multiplied by a constant, and the mean electrical axis may be accurately determined by measuring the net area bounded by the QRS deflections and the isoelectric level in two of the three limb leads. For ordinary purposes the approximate position of the mean electrical axis may be determined by inspection, except in those instances in which the net area of QRS is very small in all leads. Areas which lie above the isoelectric

level are considered positive and those which lie below it negative. If the net area is nearly zero in one lead only the mean electrical axis must be nearly perpendicular to that lead. Let us suppose that the area of R and the area of S in lead I are approximately equal, so that the net area of QRS in this lead may be considered zero. Then the mean electrical axis is at plus 90 degrees if the net area of QRS is positive in lead II and in lead III, and at minus 90 degrees if it is negative in these leads. If the mean electrical axis is perpendicular to lead II, it is at minus 30 degrees or at plus 120 degrees; in the first case the net area of QRS is positive in lead I and negative in lead III, and in the second case just the reverse. If the mean electrical axis is perpendicular to lead III it is at plus 30 degrees if the net area of QRS is positive in leads I and II, and at minus 150 degrees if the net area of QRS is negative in leads I and II. When the mean electrical axis is parallel to a given lead the net area of QRS in that lead is twice as large as the net area in either of the other leads. When the mean electrical axis is parallel to lead II it is at 60 degrees if the net area is positive in all three leads, and at minus 120 degrees if the net area of QRS is negative in all three leads. In both cases the net area of QRS in lead I will be equal to the net area of QRS in lead III and one-half as large as the net area of QRS in lead II. I need not give further illustrations; it is clear that the position on the mean electrical axis can be estimated within 30 degrees by inspection in the vast majority of cases, and that is all that is required for practical purposes.

When the heart is normal the angle which the mean electrical axis makes with the horizontal is greater than 0 and less than 90 degrees in the great majority of cases. In a few cases it falls outside these limits. In left axis deviation this angle varies from 0 to minus 60 degrees or more, and in right axis deviation from plus 90 to plus 150 degrees or more, but there is no evidence that there is a very close relation between the significance of an electrocardiogram and the magnitude of the angular displacement of the mean electrical axis which it displays.

Left axis deviation is often combined with inversion of the T deflections in lead I and right axis deviation with inversion of the

T deflections in leads II and III. In such cases the electrocardiogram is plainly abnormal and little difficulty arises in its interpretation. When axis deviation is associated with normal T waves and a normal QRS interval, the question as to whether the electrocardiogram is within or outside normal limits is not infrequently difficult to answer. When in doubt, I place more reliance upon the axis-deviation index than upon the exact amount of displacement of the mean electrical axis.

Various types of indices have been proposed. For the purpose of distinguishing between normal and abnormal grades of axis deviation, I prefer that obtained by subtracting the sum of R_3 and S_1 from the sum of R_1 and S_3, which is a function of the voltage of the chief deflections of the QRS group as well as of the position of the electrical axis. When Q is the largest of the initial deflections of lead III it should be substituted for S_3 in this formula. Table II shows that, for the elecrtocardiograms of young men, the average value of this index is —2.68, and indicates that it may be expected to exceed 14 or fall below minus 17 in about one per cent. of such curves. An index greater than plus 21 or less than minus 21 may be expected to occur in about one case per thousand.

In the electrocardiograms of the youthful conspicuous right axis deviation is not rare, but as age advances its frequency rapidly diminishes, while left axis deviation becomes more and more common. The predictions of Table II cannot logically be applied to the interpretation of the electrocardiograms of those who have reached middle age. It is my impression that when the index is greater than plus 20, the heart is almost always abnormal but an adequate statistical study might easily prove that I am mistaken.

In congenital pulmonary stenosis indices of minus 20 or less are not uncommon, but in mitral stenosis the index is seldom less than minus 15, a value which, in the first three decades of life, is by no means a certain indication of cardiac abnormality. Fortunately, this valve lesion not only gives rise to right axis deviation, but commonly produces other electrocardiographic abnormalities. It is my impression that conspicuous right axis deviation after the age of 40 is almost always abnormal.

Three examp'es of right axis deviation are reproduced in Fig. 20. The first tracing is the electrocardiogram of a very tall slender man, aged 30 years. There was a cardio-respiratory murmur, and

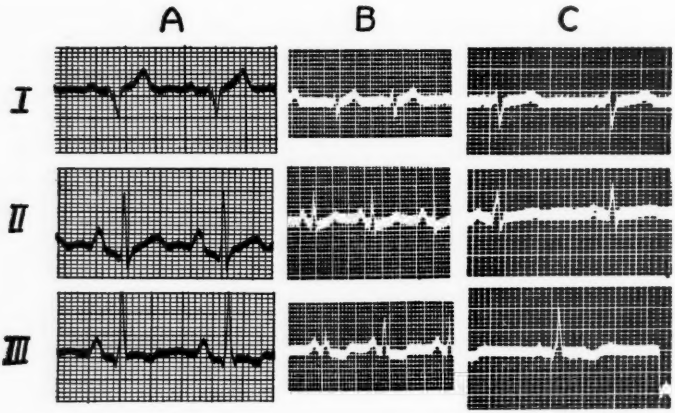


FIGURE 20. Right axis-deviation.

the first apical and the second aortic sounds were accentuated. The mean electrical axis is at plus 120 degrees, approximately, and the index is minus 20. Both of these values are outside the normal range. In addition, there is definite notching of QRS in lead I, the P waves are abnormally large in leads II and III, and the QRS interval is at the upper limit of normal. The probability that the heart is abnormal is, therefore, very great.

The second tracing is the electrocardiogram of a man aged 36 years who showed no clinical evidence of heart disease. The index is minus 4, the mean electrical axis is at plus 90 degrees, and the outline of the ventricular complex is normal. I see no reason for considering this curve abnormal.

The third tracing is the electrocardiogram of a man aged 20 years who was apparently in good health. Here the index is minus 13, and the electrical axis lies between plus 120 and plus 150 degrees. The T waves are normal and the voltage of S_1 is

not outside normal limits. The position of the electrical axis is the sole unusual feature, and I believe that no cardiac abnormality is present.

Three examples of left axis deviation are shown in Fig. 21. The first tracing is the electrocardiogram of a man, aged 51 years, who showed no evidence of cardiac disease either on physical or on X-ray examination. The index is plus 32, the electrical axis lies somewhere between minus 20 and minus 30 degrees, and S_3, which measures 15 mm., is abnormally large. According to Table II an index as large as this cannot be expected to occur in health more often than once in 100,000 observations. I should say that the probability that the heart is not normal is very great.

The second tracing is the electrocardiogram of a man, aged 56 years, whose height and weight were 5 ft. 8 in. and 197 lbs., respectively. The history and physical examination were negative.

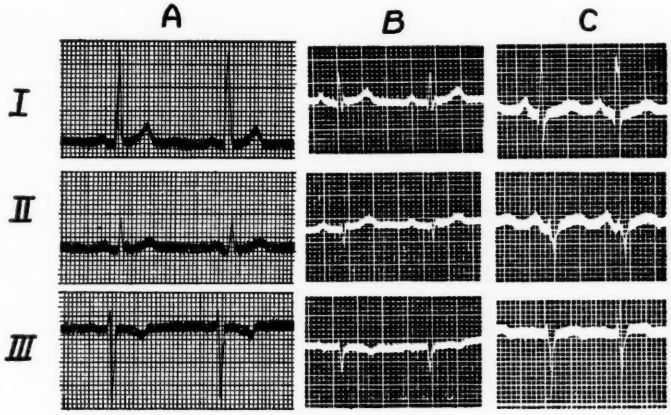


FIGURE 21. Left axis-deviation.

The index is plus 10, approximately, and the mean electrical axis is in the neighborhood of minus 60 degrees. In view of the normal index and the absence of any striking peculiarities, other than the position of the electrical axis, I regard this curve as within normal

limits. If one may judge from the height and weight, the long axis of the heart was probably nearly perpendicular to that of the trunk, and this no doubt accounts for the axis-deviation present.

The third tracing is the electrocardiogram of a man, aged 52 years, who was 5 ft. 2 in. tall and weighed 135 lbs. In this instance the mean electrical axis is at minus 60 degrees, approximately, and the index is plus 20. The S deflection of lead III measures 15 mm. This combination of values is so uncommon in health that I should hesitate to accept this curve as within normal limits.

Large Q-deflections in Lead III—In the electrocardiograms of normal subjects under 30 years of age Q_3 deflections which measure more than 4 mm. (Table II), and Q_3 deflections of which the voltage is more than one-fourth that of the largest QRS deflection in any of the standard leads [Kossman (7)] are both extremely rare. Since there is a positive correlation between the voltage of Q and the voltage of R, the largest Q_3 deflections are likely to be associated with right axis-deviation, in which R_3 is unusually large. In the electrocardiograms of persons over 40 years of age, on the other hand, large Q_3 deflections are very much more common, even when the electrical axis is in the normal position or is rotated to the left. The incidence of such deflections in angina pectoris is relatively high, and there can be no doubt that old infarction of the diaphragmatic wall of the heart, which is ordinarily accompanied by conspicuous Q deflections in lead II as well as in lead III, is sometimes responsible for the occurrence of unusually large deflections of this kind in lead III only. Nevertheless, there is no other evidence of heart disease in many of the cases in which a large Q_3 is found. Its presence under these circumstances is ordinarily ascribed to rotation of the heart brought about by elevation of the diaphragm.

The first tracing of Fig. 22 is the electrocardiogram of a man, aged 28 years, who was 5 ft. 10 in. tall and weighed 190 lbs. In this instance Q_3 measures only 3 or 3.5 mm. and is less than one-fourth as large as the R deflections of leads I and II. Although there is a Q deflection in lead II as well it is small in comparison with the R wave of the same lead and has no significance. I believe that this curve is well within normal limits.

The second tracing is the electrocardiogram of a man, aged 48 years, who was 5 ft. 11 in. tall, and weighed 198 lbs. The history and the physical examination were negative. The large downward deflection of lead III measures 6 mm. and is more than one-fourth

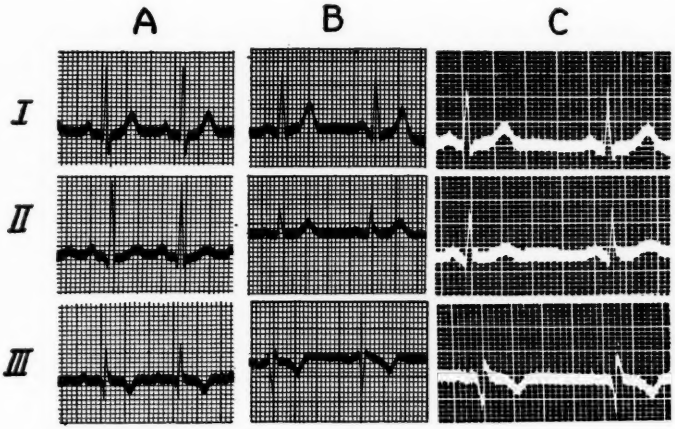


FIGURE 22. Large Q-deflections in lead III.

as large as the largest QRS deflection of the other leads. It is questionable whether this deflection should be called Q since in some complexes it seems to be preceded by a tiny upward deflection. Furthermore, both P_3 and T_3 are inverted which suggests that the angle made by the long axis of the heart with that of the trunk is unusually large. I ascribe the electrocardiographic peculiarities in this case to a transversely placed heart, and believe that no cardiac disease is present.

The third tracing is the electrocardiogram of a man, aged 44 years, who weighed 190 lbs. and was 5 ft. 9 in. tall. The history and the physical examination were negative. In this instance Q_3 measures 5 mm. and is more than one-fourth as large as R_1, which is the largest QRS deflection of any of the three leads. There is also a conspicuous Q deflection in lead II, and for this reason one must consider whether it is possible that there is an

old infarct on the posterior wall of the heart. In typical Q_3-type curves of the kind seen in posterior infarction Q_3 is larger in comparison with the largest QRS deflection, and Q_2 is larger in comparison with R_2 than in this case. Nevertheless this electrocardiogram is so much like those seen in some cases of old myocardial infarction that I should accept it as normal with a good deal of misgiving.

Precordial Leads—Leads from the precordium have not been used routinely by a large number of observers until quite recently, and it will be a long time before we shall have an adequate idea of the normal variations which they may display. For the present I can say no more than that large Q waves (more than 3 mm.), extremely small R-waves, or inverted T-waves should be considered abnormal when they occur in apical leads. Absent R-waves and inverted T-waves are probably both abnormal in all precordial leads obtained from points to the left of the left sternal margin (leads V_2, V_3, V_4, V_5, and V_6) (4). One exception must be noted; it has been reported that in childhood inversion of T in precordial leads is not uncommon when the heart is normal.

APPENDIX

For the convenience of anyone who may wish to make statistical analyses of electrocardiographic measurements, I may outline briefly the scheme employed in obtaining the statistical constants given in Table II. The cards employed have 80 columns, each of which contains the digits 0 to 9 arranged in order. The case number and the number of the lead were punched in columns 1 to 5 (see Fig. 23). The voltage of P was punched in the last columns of the next group of five, the voltage of Q in the last columns of the third group of five and so on. The case and lead numbers and the voltages of the five deflections (P, Q, R, S, and T) expressed in tenths of a millimeter or hundredths of a millivolt were entered in this way in the first 30 columns. When T was inverted a zero was punched in column 26, the first of the group of five columns devoted to this deflection. The remainder of the work was largely mechanical. By running these master cards through a multiplying

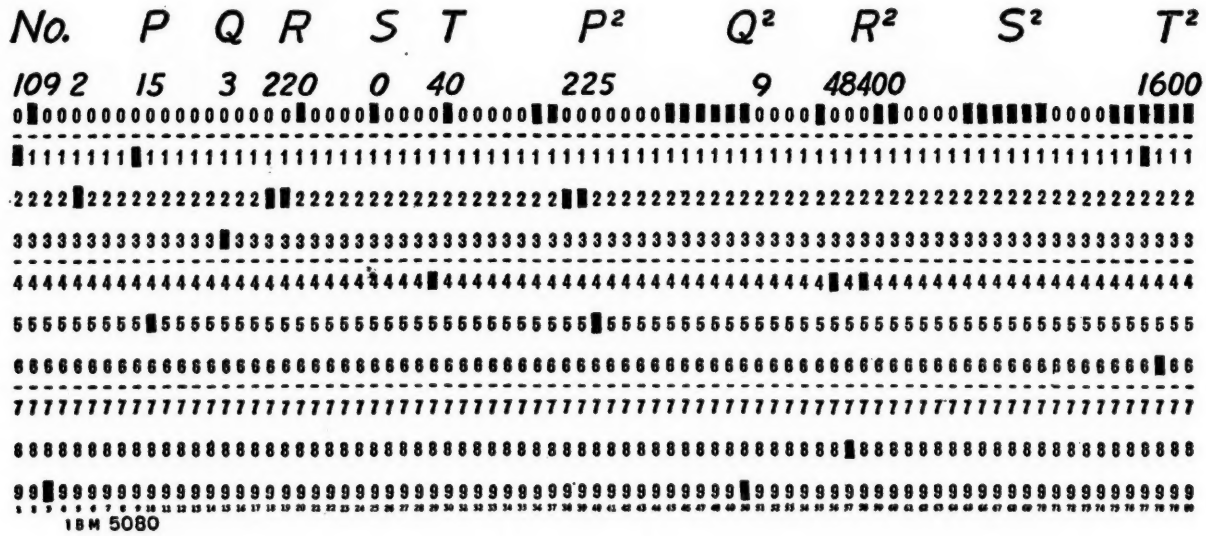


FIGURE 23. Master card used in a statistical study of the voltage of the electrocardiographic deflections.

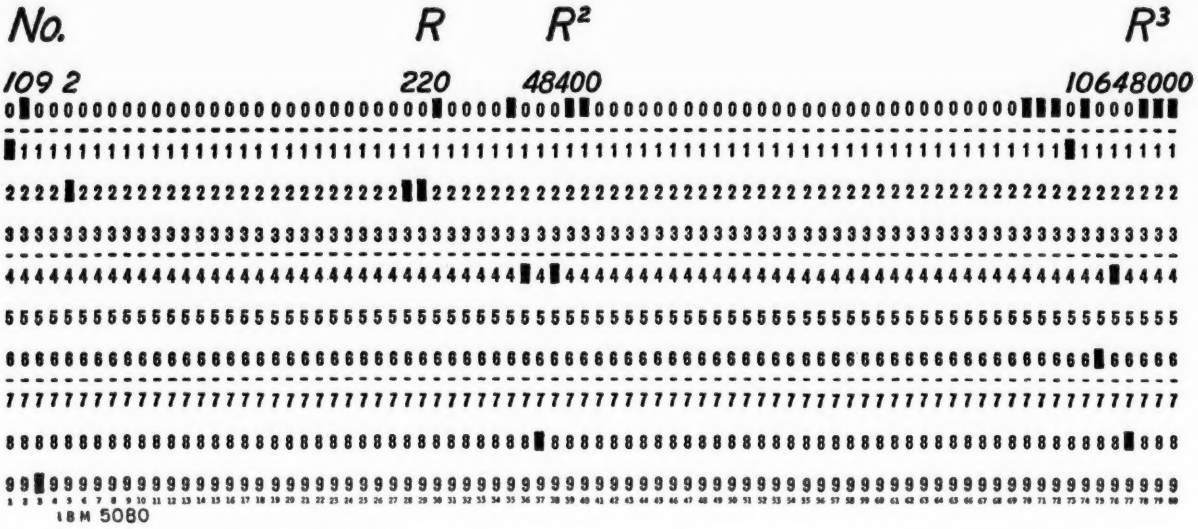


FIGURE 24. Punch card used in statistical study described in the text.

punch the squares of all of these variates were automatically punched in the last 50 columns as shown (Fig. 23). Fig. 24 illustrates the method used to obtain the cubes of the variates. A duplicating punch was used to transfer each group of variates, the squares of these variates, and the identification numbers to another set of cards. By running these through the multiplying punch the cubes of the variates were punched automatically (Fig. 24).

In order to obtain the sum of the variates, the sum of the squares, and the sum of the cubes, the appropriate cards were passed through a printer which lists, adds, and prints the sums in one operation.

The rest of the work was done with a Monroe calculator by utilizing the following equations.

$$\text{Mean} = \frac{\Sigma v}{N}$$

$$A = N\Sigma v^2 - \Sigma v \Sigma v$$

$$B = N\Sigma v^3 - \Sigma v^2 \Sigma v$$

$$C = NB - 2A\Sigma v$$

$$\text{Standard Deviation} = \frac{1}{N\sqrt{A}}$$

$$\text{Skewness} = \frac{C}{A\sqrt{A}}$$

N = Number of variates.

Σv = Sum of the variates.

Σv^2 = Sum of the squares of the variates.

Σv^3 = Sum of the cubes of the variates.

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Dr. Brown—You will recall that Dr. Wilson, at the start of his address, very graciously gave you permission to leave if you became tired, bored or uninterested. The fact that there is a greater attendance now than at the start of his talk may be taken by Dr. Wilson as an indication of the interest which has been taken in his paper and as an index of the excellence of the material which he has presented.

Many of us in New York and vicinity have come to know a very likable physician who, because of very hard and intensive work and a native ability, has acquired an enormous knowledge of the organs of circulation. Many of us have depended in a large degree upon his diagnoses in accepting some risks for insurance and in acknowledging or denying a heart claim for disability.

He is Professor of Therapeutics, Department of Medicine, New York University; member of the Criteria Committee, New York Heart Association; Medical Director, Irvington House for Cardiac Children; and Consulting Cardiologist to several hospitals. It is both a pleasure and a privilege to have Dr. Arthur C. DeGraff discuss the discourse to which we have just listened.

Dr. DeGraff—I don't know that I can quite live up to all the things that Dr. Brown has said of me. However, I will be very brief, I assure you, in my discussion.

Dr. Wilson has so adequately covered the subject that there is really very little for me to add. I should like to say something, however, about the normal series that Dr. Wilson suggested should be made.

In my contacts with insurance work, the need for such a series is certainly definitely evident. A few years ago, on our own initiative, we tried to make a study of normal electrocardiograms in medical students. We met the same difficulties that Dr. Wilson encountered and which he gave as the reason for not doing the work at his place: The work was difficult, time-consuming and we did not have enough money to carry on very far. We did, however, study two hundred students, taking several electrocardio-

grams in each case. Some of that work has been published. The need for thousands of cases is certainly very evident.

The question of standardization of nomenclature is very important. Those of us who are working on the Criteria Committee of the New York Heart Association have realized for a long time the necessity of a standardized nomenclature for ordinary heart work. This is even more important for insurance work because, unless people talk the same language, there is always confusion.

One of the important difficulties at the present time is the naming of the *Q* wave. I know that some people refer to a small initial upward deflection as a *Q* wave. In the nomenclature of the American Heart Association, only an initial downward deflection is considered a *Q* wave. At the present time, we consider that an initial downward deflection is a *Q* wave and a final downward deflection is an *S* wave and an upward deflection is an *R* wave. Anything else in between is not named. The whole situation does need some clarification and I think that certainly the insurance work on normals will help us a great deal.

I should also like to say something about slurring of the *R* and *S* waves. In disability work particularly, even slight slurring of either of these waves is considered by a number of doctors as definite evidence that the man has serious heart disease and should be entitled to total disability benefits even though there are no other deviations from normal in the electrocardiogram. Certainly a great many times slurring of the *R* wave or of the *S* wave is related to the thickness of the string of the galvanometer. The greater the deflection, the less slurring there will be. If there is a small deflection, there will be slurring in practically every case. Slurring then is certainly not abnormal.

Just a word about the method that Dr. Wilson has described for chest leads. A few years ago, I began to use this method and have found it extremely valuable. It is not time-consuming. It takes only three minutes longer than the ordinary, so-called lead 4. It is much more satisfactory, I find, because it uses several points and it gives us not just one chest lead which might be equivocal but several leading-off points, enabling us to decide more definitely whether or not an abnormal situation exists. It is not necessary

in most cases to use a single stage amplifier such as Dr. Wilson has in his circuit. If the resistance is low, it can be dispensed with.

We all appreciate the amount of work that Dr. Wilson has done in this field. We appreciate the value of his presentation this afternoon. His talk will help a great deal in evaluating certain features about the electrocardiogram; but more than that, it has made clear to us that much work still must be done in this important field.

DR. BROWN—Thank you, Dr. DeGraff.

* * * * * *

DR. BROWN—Blood pressure is an old subject in life insurance. Without life insurance examinations, blood pressure would doubtless not have been the popular subject of conversation with the laity which it has become. Here is one instance in which the clinician took a leaf from the book of the medical insurance worker.

Dr. Lewis F. MacKenzie and Mr. Pearce Shepherd, of the Prudential Insurance Company of America, have recently done some investigating and embodied their results in a paper entitled, "The Significance of Past Hypertension in Applicants Later Presenting Normal Average Blood Pressure." Mr. Shepherd, will you give us an outline of your findings?

THE SIGNIFICANCE OF PAST HYPERTENSION IN APPLICANTS LATER PRESENTING NORMAL AVERAGE BLOOD PRESSURES

LEWIS F. MACKENZIE, M. D., *Associate Medical Director,* AND PEARCE SHEPHERD, *Assistant Actuary,*

The Prudential Insurance Company of America

Until recently, blood pressure mortality investigations have been concerned with a determination of the mortality for different degrees of elevation in the current pressure. The realization has been growing that there are factors other than the current pressure which are partly responsible for the very high mortality ratios brought out in these studies. One of these factors is a history of previous hypertension. This paper presents an experience aimed at measuring the importance of a previous finding of high blood pressure in risks later showing a lower pressure.

The study is confined to individuals who applied to The Prudential Insurance Company of America during the years 1920 to 1935, inclusive, and were granted standard insurance. All blood pressure determinations are, therefore, those found on examination for life insurance. All entrants were presumably free from other impairments since they were considered eligible for standard insurance. Current diastolics were supposed to be taken at the 5th phase, but we cannot be positive about the reports based on the earlier examinations, as the 4th phase may have been used in some instances.

There are many cases where there is only one high blood pressure reported prior to our examination, with no other findings intervening. There are others where there have been numerous findings over a period of years, sometimes with many low readings following one or more high ones. We have made no distinction between these two types in the studies that follow.

During the earlier part of the period covered, a slightly elevated blood pressure was regarded more leniently than it has been in more recent years. This is fortunate for our purpose, for it enables us to study a broader range of current readings than would otherwise be possible. Until recent years, no formal rule has been in effect regarding the significance of a high blood pressure in the past, with the result that such cases were rather freely accepted.

The conviction that such past records could not be regarded lightly led to the adoption of a procedure which attaches considerable importance to a previous high record, as will be clear from a statement of our present rules. A "current blood pressure" is determined as the average of all different-day readings obtained. If several readings are obtained on one day, the average of these, disregarding, of course, those obtained immediately following exercise, is taken as that day's determination. The highest systolic and the highest diastolic on record, not necessarily obtained at the same time, is taken as the "previous record". The blood pressure rating table is entered with the mean of the current and previous systolic pressure and the mean of the current and previous diastolic pressure. The table referred to may be found in Volume 34 of the Transactions of the Actuarial Society, page 296.

The cards used had been written over a period of years, but it was necessary to supplement the information originally put on the card with further details obtained by reference to the application papers in a large percentage of the cases. We can say, therefore, that the current and previous pressures used in this study are those described in our present rule except in a small percentage of cases which is not of sufficient weight to appreciably affect the results. There were some files which could not be obtained. This data which could not be classified is shown as "N. S." (Not Stated). A complete classification would make only minor changes in the ratios.

Standard issues of 1920 to 1935, inclusive, were followed to the end of 1936. The experience is by policies. After considerable thought it was decided to calculate the expected deaths by the use of the Basic Table 1920-1934 which had been prepared for use in

the Impairment Study of 1936 made under the direction of the Joint Committee on Mortality. It was necessary to extend the table to cover the longer durations present in our study.

We adopted this table with some hesitation because we believe it is not an accurate guide to normal mortality *by policies* over this period. Unfortunately, we cannot prove our contention conclusively, but we would like to offer our reasons for believing as we do.

We all recognize that a true mortality investigation should be made by *lives*. This is troublesome to do and as an approximation it is customary to assume the experience by *policies* will closely parallel that by lives. Quite often it is equally valid to assume that the experience by amounts of insurance will parallel that by lives and by policies. Mortality rates on all three bases will be identical as long as the relation between lives, policies and amounts is the same among the deaths as among the exposed. If the relation is not the same, the rates will differ. A relatively high mortality on policies of large amounts, such as has been experienced in recent years, would give a higher mortality rate by amounts than by policies.

Studies of broad groups by both *policies* and *amounts of insurance* in our own company and in other companies indicate that there is comparatively little difference in the rates on these two bases except at ages 35 or 40 up to 60. Over this period the rate by amounts may be as much as 20% more than the rate by policies. It is possible, therefore, that the table in question *overstates* the expected deaths on a policy basis by perhaps 15% at ages 40 to 60 or over, so that our ratios are too low. The younger ages are also affected at the longer durations.

We have not attempted to modify the table or the results in any way, but we feel this word of caution in interpreting the results is necessary.

The results of the study by systolic pressure are given in some detail in Table I.

At ages under 30 the mortality is low, but the volume is too small to be conclusive.

At ages 30-39 there is a larger volume exposed, but the bulk of the experience is on issues at ages 40-59. The mortality on issues at ages 60 and over is comparatively favorable, with a sizeable number of deaths. An adequate explanation of this favorable experience is not readily apparent. It cannot all be accounted for by the fact that pressures average higher at these ages. Perhaps it is due to the ability of underwriters to sense more than appears on the surface and thus pick only the best of the applicants at these ages. Perhaps our standard table indicates too high a death rate at these ages. Whatever the reason, it seems best to confine further detailed study to ages at issue 30-59.

Table II combines some of the groups shown in Table I. It is clear that the high mortality shown is a function of both the current and the previous pressures. Even with current systolics under 135 mm., there is a substantial excess mortality, the amount of the excess depending on the height of the previous pressure. Within a given range of previous pressures, the mortality increases with increased current pressure, this increase being more than can be accounted for by the difference in current pressures alone.

It appears from this study that a pressure of 150 mm. or more in the past requires a rating, even though current pressures are within normal limits. A previous record of 165 mm. or over with moderate elevation on examination requires a substantial rating or rejection.

We have looked into the effect of duration since issue of insurance. The figures show a fairly constant extra mortality. The standard table might easily be misleading in this phase of the study and we would not be surprised to find that the mortality increases with duration when measured by a more suitable standard.

We also separated those cases where the previous reading was more than two years prior to our examination, from those where it was within two years. Apparently it makes little difference although this does not mean that there may not be some division of the data along these lines that will yield significant results. It does, however, tend to discourage the discounting of a previous record on the strength alone of the time since it was reported.

How does the mortality of the group with a previous high pressure compare with that of a group showing similar pressures on examination? Putting it another way, how much credit (against the rating for the high pressure) is justified on the strength of a normal or near normal current pressure?

A parallel experience dealing with current pressures is needed to answer this question definitely, and such an experience should exclude cases with a previous high reading. In the absence of such an experience we can compare the rating for current pressures with the roughly graduated ratios indicated by the present study. Such a comparison indicates that cases with previous records call for ratings only slightly lower than cases which show current correspondingly high pressures. A rating based on the mean of the current and previous is too lenient; a rating which is the mean of the *ratings* for the two is better but is still not adequate. Apparently at least twice as much weight should be given to the previous high reading as is given to the current reading.

Although a little aside from the main theme, we should like to express the opinion that there is no reasonable justification for considering a risk with current hypertension more favorably because of previous lower pressures.

We attempted a study on the basis of previous diastolic pressure regardless of systolic without very satisfactory results. Some cases are included in this study that were excluded from the systolic study because that pressure was under 135 mm. The experience for ages 30 and over is given in Table III.

It is physiologically correct to assume that increase in the diastolic pressure develops some time after an increase in the systolic begins. Recent studies have demonstrated that mortality is a function of both and that a knowledge of each is necessary for a correct interpretation and estimation of the risk.

The prognosis of diastolic readings of 90 mm. and over is largely dependent on the height of this pressure and the height of the accompanying systolic pressure. A definitely high diastolic, say 95 mm. or over, with a low systolic suggests that the systolic has been higher and is a danger signal that the heart is failing under

its increased load. When both diastolic and systolic are high, we know that the heart is seriously overburdened and may be approaching the breaking point.

We have not classified the present material using both pressures because we felt that the volume would not justify this refinement. The relation has been so clearly shown in the case of current pressures that there can be no doubt that the same applies to this material. Specifically we would expect the group with a previous systolic of 165 mm. to be divisible into groups determined by the previous diastolic and that the mortality would be highest when a previous systolic of 165 mm. was associated with a previous diastolic of the highest order.

In spite of all the reasons which support the thesis that the diastolic is more important than the systolic, the latter appears to have some statistical advantages. Perhaps further analysis of the material will show the diastolic to better advantage.

It is customary in studies such as this to analyse the causes of death. We have used as a measure of the normal deaths that might be expected, the same table that was used in the Impairment Study of 1936. Such a comparison can only be approximate, but there can be no denying the significance of the great number of deaths in just those groups which might be expected to account for the extra mortality. The results are shown in Table IV for the material included in the systolic study.

It is quite possible that not all the individuals entering our study had essential hypertension. In some cases there was only one record of high blood pressure prior to our examination, but there were many in which there were several normal or nearly normal readings taken subsequent to the high finding but prior to our examination. We consider it safe to conclude, therefore, that the majority of our cases did have essential hypertension, for all were thoroughly investigated prior to the granting of standard insurance.

May we say that essential hypertension is not a disease, but is rather a sign of circulatory change which may appear only occasionally in the early stages. Its significance is related to the extent of the departure from the normal pressure for the age. That its

seriousness is not altered by occasional low pressures, is well illustrated in the present study. It is not, so far as we know, primarily due to any circulatory impairment but is provocative of it. A sign so suggestive as this of probable serious change in both the heart and arteries, even though it be moderate on examination, certainly calls for searching investigation of the circulatory system, one or more specimens of urine to the Home Office and greater care, if possible, in the selection of examiners on whose reports and opinions we base our decisions. If this is thought to be too thorough, exacting or expensive a procedure, a glance at the proportion of cases in our study terminating in cardio-vascular-renal deaths should dispel any doubt. (Table IV.)

While we are not yet ready to report on the influence of family history on the mortality of our groups, we feel that this feature warrants our consideration. Man's structural make-up and future functional normalcy are largely determined prior to birth. He enters life blessed or cursed by his ancestry. Heredity is, therefore, vitally important, as through it the various organs and tissues are endowed with strength to resist or predisposition to develop certain types of impairment.

Both the nervous and circulatory systems are particularly subject to the influence of heredity. We cannot escape the conclusion that the inability of certain portions of these systems to withstand adequately the daily stress and strain to which they are subjected, to resist excessive influences imposed through malfunction of one or more of the endocrine glands, or the deleterious products of faulty metabolism, is frequently inherited.

We are aware that actuarial investigations of the effect of family history have in many cases tended to minimize the importance of this feature. There have been few attempts, however, to measure its effect when coupled with a related slight defect in the applicant. We are hopeful that studies of our material may enable us to separate cases with a predisposition to cardio-vascular-renal disease from those without this predisposition and thus suggest more equitable treatment.

The above comments on heredity and family history indicate a very definite bearing on the possible origin, rate of progress and prognosis of essential hypertension which *per se* is not a disease, but is provocative of it. Its insidious and symptomless beginning is fraught with grave danger to its victim who is usually quite unaware of its presence, at least until symptoms of impaired circulatory function lead him to seek medical advice, or until it is discovered at a health or insurance examination.

Our present thought is that it usually has its beginning in an imbalance of the autonomic nervous system. The amazingly intricate adjustments of this part of the nervous system become increasingly upset and its control of the heart and blood vessels progressively disturbed. Just what part disturbances of the endocrine glands, particularly the adrenals, pituitary and thyroid, take in this imbalance does not appear to be sufficiently clarified to justify detailed discussion at this time. There is a possibility that one or more of them may be a primary cause. That they are an important factor seems probable.

The earliest indication of this imbalance, such as "nose-bleed, menstrual disturbances, flushing, migraine, palpitation and nervousness" (Clinical Heart Diseases, S. A. Levine, 1936, page 157) may be present prior to puberty or during adolescence. Too little effort has been made to ferret out such histories in applicants suspected of hypertensive tendencies. An attempt to this end, even though unrewarded at times, might in many instances furnish important clues as to probable future developments and prove to be of material assistance in prognosis.

To disregard temporary rises in blood pressure as merely the result of emotion, nervousness or excitement at the time of examination is to overlook the true import of the findings. Any rise in systolic pressure (over 15 mm. above the average for the age) should be looked upon as indicating a potential or actual case of hypertension.

The imbalance of the autonomic system means the arterioles constrict and the heart is called on to expend more energy. This response leads to increasing and more oft repeated insults to the

heart and arteries. Thickening of the walls of the arterioles and cardiac hypertrophy occur in the majority of cases. Because of the thickening and increased tone of these vessels peripheral resistance is increased, adding an additional burden to the already overworked heart. When this occurs a vicious circle has been established between the nervous, circulatory and, possibly, endocrine systems resulting in generalized arteriolar change with localizing effects in one or more of the coronary, renal, cerebral, splenic, hepatic and other vessels, ultimately resulting in degenerative changes in the organs affected and, as time goes on, in myocardial breakdown.

The auscultatory gap is to be remembered as a source of danger. It is not infrequently present in hypertension and may be very misleading, unless care is taken to raise the pressure at least 20 mm. above the point where the sound is lost as the cuff is being inflated. We quite often meet with cases reported as having normal systolic pressures, but with previous evidence of hypertension. Additional readings, more carefully taken, again show its presence.

A very brief and necessarily incomplete attempt has been made to explain the more outstanding reasons for the high mortality ratios shown in the attached tables.

One thing appears to be certain, namely, that essential hypertension once begun is incurable. Too few realize that in its beginning and for long periods it may be present only occasionally, but when detected it is a danger signal that must not be made light of or disregarded by either its victim or the insurance company to which he may apply. It is positive evidence of a probable shortened span of life. The fact that some live for years with enormously high pressures does not detract from the above statement.

Because of the significance of the mortality ratios here presented, we wish this to be regarded as a preliminary report, which we hope to supplement at a later meeting. Our intention is to review the data, here shown, in much greater detail and to add to it so that the increased number of cases and the longer exposure may lend added weight to our deductions and conclusions.

TABLE I

Current Systolic Pressure

Age at Issue	100-124 mm. Deaths	100-124 mm. Ratio	125-134 mm. Deaths	125-134 mm. Ratio	135-144 mm. Deaths	135-144 mm. Ratio	145 mm. & Over Deaths	145 mm. & Over Ratio
Previous Systolic 135-144 mm.								
–29	6	159%	1	26%	2	171%	....	....%
30–39	6	161	3	54	6	159	....	....
40–49	2	61	9	140	6	92	....	....
50–59	1	22	7	159	4	60	....	....
60 & over	0	0	1	32	6	118	....	....
Previous Systolic 145-154 mm.								
–29	3	118	1	28	2	101	0	0
30–39	4	103	19	221	13	201	2	339
40–49	10	176	25	191	39	177	12	277
50–59	7	167	21	152	62	196	16	157
60 & over	4	301	4	105	19	109	7	141
Previous Systolic 155-164 mm.								
–29	1	92	1	59	0	0	0	0
30–39	3	155	2	50	10	189	0	0
40–49	5	139	10	114	46	265	20	323
50–59	7	149	20	162	45	147	28	155
60 & over	2	426	2	48	22	170	22	152
Previous Systolic 165-174 mm.								
–29	0	0	0	0	0	0	0	0
30–39	0	0	0	0	0	0	0	0
40–49	0	0	9	265	9	194	3	224
50–59	2	213	6	173	30	231	22	300
60 & over	0	0	2	123	11	141	6	195
Previous Systolic 175 mm. and over								
–29	0	0	0	0	0	0	0	0
30–39	0	0	0	0	1	145	0	0
40–49	0	0	1	82	9	386	2	333
50–59	0	0	6	345	20	342	9	153
60 & over	0	0	1	60	2	55	0	0
Previous Systolic Not Stated								
–29	0	0	0	0	0	0	0	0
30–39	4	280	3	330	3	345	0	0
40–49	5	218	7	246	4	178	0	0
50–59	4	317	1	49	4	110	3	167
60 & over	0	0	1	217	0	0	0	0
All Previous Systolics								
–29	10	122	3	30	4	92	0	0
30–39	17	150	27	133	33	180	2	140
40–49	22	137	61	170	113	205	37	272
50–59	21	127	61	162	165	180	78	180
60 & over	6	147	11	74	60	120	35	131
ALL AGES	76	135	163	137	375	171	152	179

TABLE II

Current Systolic Pressure

Previous Systolic	100-134 mm. Deaths	100-134 mm. Ratio	135-144 mm. Deaths	135-144 mm. Ratio	145 mm. & Over Deaths	145 mm. & Over Ratio
Ages at Issue 30–39						
135–144	9	97%	6	159%	....	.....
145–164	28	152	23	195	2	213%
165 & over	0	0	1	53	0	0
Ages at Issue 40–49						
135–144	11	114	6	92	....	.....
145–164	50	160	85	216	32	304
165 & over	10	172	18	258	5	258
Ages at Issue 50–59						
135–144	8	90	4	60	....	.....
145–164	55	157	107	172	44	156
165 & over	14	198	50	266	31	234
Ages at Issue 40–59						
135–144	19	102	10	76	....	.....
145–164	105	159	192	189	76	196
165 & over	24	186	68	263	36	237
Ages at Issue 30–59						
135–144	28	100	16	94	....	.....
145–164	133	157	215	189	78	197
165 & over	24	166	69	249	36	231

TABLE III

Experience by Previous Diastolic Pressure

Previous Diastolic Pressure	AGES AT ISSUE 30-39 Deaths	30-39 Ratio	40-49 Deaths	40-49 Ratio	50-59 Deaths	50-59 Ratio	60 & Over Deaths	60 & Over Ratio
– 90	56	120%	87	111%	117	144%	46	105%
91– 95	6	111	11	79	35	186	5	50
96–100	15	147	65	216	90	192	27	150
101 & over	18	224	75	301	92	154	30	135
N.S.	12	230	25	220	18	151	7	99
All	107	142	263	166	352	161	115	114

TABLE IV

Analysis by Cause of Death

Previous Systolic Pressure 135 mm. or more

Cause of Death	Ages 15-39 Actual	Ages 15-39 Expected	Ages 15-39 Ratio	Ages 40 & Over Actual	Ages 40 & Over Expected	Ages 40 & Over Ratio
Cardio-Vascular-Renal: Apoplexy, Cerebral Hem., Embolism, etc.	5	.8	617%	61	40.6	150%
Heart Diseases	22	5.4	408	261	83.5	313
Nephritis	16	2.1	773	77	20.3	380
Total	43	8.3	518	399	144.4	276
Tuberculosis	5	11.1	45	11	16.2	68
Cancer & other malignant tumors	0	3.5	0	76	51.1	149
Pneumonia	13	6.2	210	42	34.5	122
Suicide	9	3.3	277	28	16.2	173
External causes other than suicide and homicide	7	14.8	47	30	27.2	110
All other causes	19	26.7	71	84	116.0	72
ALL CAUSES	96	73.8	130%	670	405.0	165%

Dr. Brown—Dr. John M. Livingston, of the Mutual Life Assurance Company of Canada, will open the discussion.

Dr. Livingston—It would seem a truism that the less a subject is understood the more is written about it and this may apply to essential hypertension. Theoretically, essential hypertension may be attributed to many factors but the evidence now seems to be quite clear that actually there is an increase in the peripheral resistance produced by an unknown cause.

There is no clear evidence that there is an increase in a circulating pressor substance in the blood which might produce hypertension. If any such substance, either alimentary, metabolic or endocrine, were responsible in nature for the heightened blood pressure, one should expect that a pressor effect of the serum could be demonstrated in subjects showing hypertension. Attempts to do so have failed. The role of infection and allergy in the aetiology have been studied without any definite results. Definite evidence has been produced that the carotid sinus plays no part in the production of essential hypertension and there is little real

evidence that changes in the medullary vessels bear a primary relationship to the condition.

It is known that in essential hypertension there is an abnormal response of the arterioles to stimuli. Whether this results from abnormal vasomotor impulses from the brain or whether the arterioles react abnormally to normal stimuli is not of great importance. There is some evidence that there are no abnormal vasomotor stimuli in essential hypertension and we may assume that the arterioles react excessively to normal stimuli. There appear to be two factors in the elevation of blood pressure in hypertension: (1) abnormal reaction of the arterioles to stimuli and (2) the inherent status of the arterioles. The abnormal response is probably the dominant cause in cases showing wide fluctuations of blood pressure. When the blood pressure is high and fluctuates little it is probable that the inherent or automatic function of the arterioles is dominant. This automatic function of the arterioles may result, in part, from organic changes, which have been shown to exist in increasing degree as the severity of the hypertension increases.

The blood pressure usually fluctuates widely in essential hypertension and under the stimulus of emotion or cold it increases greatly. Hines & Brown have shown by means of the "cold test" that hypertension can be invoked transiently in a very small percentage of individuals who have normal blood pressure and when family histories do not disclose hypertension. When there is a family history of hypertension, a positive response to the "cold test" is found in a much larger percentage of instances, so it appears that in certain individuals there is a hereditary susceptibility to hypertension. A study of family history in this group may give results of note.

Table II shows, for ages at issue 30-59, that if the current and previous systolic pressures are under 145 mm. the mortality is quite favorable and one may feel that such cases may be accepted for standard insurance. However, when pressures are, *or* have been found, over 145 mm., there is a rather marked increase in mortality and yet such a dividing line does not seem reasonable. The age group 40 to 49 years, as in previous studies, shows the

worst mortality and yet there appears to be no adequate explanation of this experience.

It would be of interest to know what percentage of these applicants would have been considered substandard under the revised procedure now in use and the present table for blood pressure ratings. I should judge, however, that, if the revised procedure and present table of ratings had been used, the actual mortality would have been definitely greater than the expected, for it is stated: "A rating based on the mean of the current and previous is too lenient; a rating which is the mean of the rating for the two is better but is still not adequate. Apparently, at least twice as much weight should be given to the previous high reading as is given to the current reading."

I was interested particularly in the group showing a current systolic blood pressure of 145 mm. or over, and a previous higher current pressure. This group undoubtedly was very carefully selected when it was granted standard insurance. I have prepared a table for this group which, for exposure 1-17 years and ages 30-59, shows a mortality of 206% and shows a definite increase in mortality by duration.

I agree with the essayists that we should disregard the frequent excuses for a temporary rise in blood pressure, that it was due to emotion, nervousness or excitement at the time of examination. The conditions and circumstances of a properly conducted insurance examination do not allow such excuses to be considered a satisfactory explanation.

In actual practice I think it will be difficult to give the same weight to previous high pressures recorded five years ago as to those found more recently. Similarly one previous high record will cause difficulties, especially when we have no knowledge of the examiner who made the estimation. Practically all other impairments take note of time elapsed and number of attacks.

It is disappointing that study of the diastolic pressures did not show definite results. From a physiological and pathological viewpoint, it should help in separating those in the second degree of hypertension. I have noticed quite a number of death claims from cardiovascular disease in the first ten years and yet, on

TABLE I

CURRENT SYSTOLIC PRESSURE 145 MM. AND OVER
AND
PREVIOUS SYSTOLIC PRESSURE 145 MM. AND OVER

Years	Age	Deaths	Rate
1– 5	30–39	0	0
	40–49	10	208
	50–59	23	119
	60 & over	15	125
	All ages	48	131
	30–59	33	134
6–10	30–39	2	339
	40–49	15	285
	50–59	42	239
	60 & over	18	168
	All ages	77	225
	30–59	59	252
11–17	30–39	0	0
	40–49	12	502
	50–59	10	222
	60 & over	2	66
	All ages	24	235
	30–59	22	307
1–17	30–39	2	144
	40–49	37	297
	50–59	75	181
	60 & over	35	136
	All ages	149	184
	30–59	114	206

reviewing the papers, the only item of note is a diastolic blood pressure of 90 to 95 mm. (fifth phase). I am hopeful that further study will give more definite information of the significance of diastolic blood pressure.

There are many different tables in use for rating blood pressure and they vary greatly. There are probably a greater number of different procedures used in determining the blood pressure to be considered as applicable to a given case when several estimations of blood pressure are available. If a particular table is used with a procedure other than that recommended for that table, many very bizarre results are obtained. Reasonable uniformity in procedure probably will be necessary before we can have uniformity in decisions. Information is accumulating gradually which should allow us to give proper consideration to the higher

readings and to a history of blood pressure. To this end Dr. MacKenzie and Mr. Shepherd in this study have made a valuable contribution.

DR. BROWN—Dr. William Bolt, of the New York Life Insurance Company, will continue the discussion.

DR. BOLT—The present investigation is interesting to me because it covers much the same ground as our New York Life investigation in 1934. It was our idea that a past history of hypertension was just as important as a past history of tuberculosis, gastric ulcer or any other serious impairment, and that low and normal blood pressures which are obtainable in most hypertension cases under favorable conditions should not be allowed to offset previous high readings. The present Prudential investigation deals with issues of 1920 to 1935 carried to policy anniversary in 1936, the bulk of the material from which conclusions are drawn being from ages 40 to 59. The New York Life study in 1934 covered the age period from 40 years on, with issues of 1923 to 1928 carried to policy anniversary of 1933. The methods followed in the two investigations were somewhat different, the Prudential taking into consideration the highest systolic and the highest diastolic on record (the two not necessarily being in the same reading) and the current blood pressures by averaging all blood pressures found at the time of examination. The New York Life study covered (1) the single highest systolic and single highest diastolic and (2) the average of the two highest systolics and the two highest diastolics (not necessarily in the same reading).

I agree with Mr. Shepherd that the term "average blood pressure" is merely a convenient device for comparison purposes. We must have some standard with which to make comparisons and the average blood pressure is useful for this purpose. We know, however, that a man's blood pressure will vary considerably from day to day or at different times during the same day. It is doubtful, therefore, whether the plan of averaging several blood pressures obtained within a short time is of much practical value.

I am rather sorry that the authors have included in the report some material subdivided into many small groups. This results

in a high mortality ratio based on a very small number of deaths —something that often leads to seeming inconsistencies. Thus, in Table I, in the second subdivision dealing with previous systolic pressures 145-154, current systolic pressure 145 and over, in the age group 30 to 39, there were only two deaths, with a mortality ratio of 339. In the last subdivision of that same Table I, including all previous systolics in the same age group and the same current systolic range, there are still only two and, presumably, the same two deaths, and yet the mortality ratio is 140. Again, if we take the group where these two deaths give a mortality of 339, we find that this mortality ratio is much higher than in the fourth subdivision where the age group is older; i. e., 40 to 49, and the previous systolic is higher; i. e., 165-174. In this latter group there is a mortality ratio of 224 based on three deaths. It might have been better to have avoided results of this sort by fewer subdivisions of the material. Of course, the authors point out that the bulk of their experience is from cases aged 40 to 59, where the groups are larger.

Table II is interesting and the second last subdivision, comparing the result for ages 40 to 59, shows some similarity to the New York Life investigation of 1934, as indicated below.

TABLE II

PRUDENTIAL

Age 40 to 59

Previous Systolic	Current Systolic 100-134 mm. Ratio	135-144 mm. Ratio	145 mm. & over Ratio
135–144	102%	76%	.....
145–164	159%	189%	196%
165 & over	186%	263%	237%

NEW YORK LIFE

Age 40 and Over

	Sys. or Dias. Synthetic Blood Pressure	Ratio
Standard & Substandard	150–154 / 100–101	117%*
	155–159 / 102–103	144%
Substandard	160–164 / 104	186%
	165–169 / 105	236%
	170–174 / 106+	249%

*Combination standard group Table VIII and substandard group Table IX.

In general, the results of the Prudential investigation are similar to our conclusions based on the New York Life study in 1934. Both investigations indicated that an extra mortality is to be expected where the blood pressure has at some time been found elevated. The Prudential investigation concludes that an extra mortality is to be expected if the systolic exceeds 150 and that a substantial rating or declination is necessary where the systolic has reached 165. In our investigation we found that, if the systolic had not exceeded 150 at any time and there were no other unfavorable factors, a favorable experience may be anticipated. The Prudential investigation also found the same as we did: that the time elapsed since the previous unfavorable readings appeared to make no difference in the experience and, moreover, that it was not wise to accept the explanation that a previous high reading was due to emotional stress or excitement. This is reasonable because an individual whose nervous system is so constituted that he shows a rise in blood pressure when being examined is probably the type of individual who becomes unduly upset when he meets other episodes disturbing to him in the course of his daily work.

The Prudential found that approximately 60% of the deaths over age 40 were due to cardiovascular-renal disease. This is a somewhat lower average than we found, as about 73% of the deaths in our group were due to some degenerative disease.

In regard to the rating of cases where there is a history of hypertension but normal or nearly normal blood pressure on examination, the authors suggest that apparently twice as much weight should be given to the previous high reading as is given to the current reading. We are inclined to believe that the rating should be based on the high readings, with little or no credit for current more favorable readings. This view we believe is justified by the fact that the authors of this paper found that a comparison indicates that cases with previous records called for ratings only *slightly* lower than cases which show current corresponding high pressure. Our New York Life experience shows that the mortality is proportional to the high readings.

Certainly the information derived from these two independent investigations clearly warrants careful consideration of a past history of hypertension in medical underwriting and indicates that those who ignore such a history must be prepared for an unfavorable experience.

I believe, too, that we should spread this information around among practicing physicians as much as possible. There are still too many clinicians who underestimate the danger of a moderate elevation of blood pressure, particularly if a few simple measures suffice to bring the blood pressure back to a lower level. It is probable that those of us in insurance medicine see more evidence of early cardiovascular-renal disease than the average physician. Too many of these cases come to their doctors when it is too late to do much good. If we disseminate our knowledge as widely as possible and whenever opportunity offers, we may in time help to prolong many lives.

I wish to thank Dr. MacKenzie and Mr. Shepherd for the privilege of discussing their paper.

DR. BROWN—Our schedule will allow us to devote a little more time to this subject. If there is anyone in the audience who would like to continue with this discussion, we should like him to feel privileged to do so.

DR. WILLIAM E. FERGUSON (Excelsior Life Insurance Company, Toronto, Canada)—There is one point I should like to comment upon. I have run into a few instances where people, who have had impairment of blood pressure reported against them, ran a very favorable blood pressure when examined; but where we had experienced examiners and we sent such examiners back, when no arrangements had been made and the man was not aware that the blood pressure was going to be rechecked, we got substantially higher readings.

DR. BROWN—Is there anyone else who has a comment? If not, I will ask the other author of this paper, Dr. MacKenzie, to close the discussion.

Dr. MacKenzie—I first wish to express my thanks and appreciation, and I am sure Mr. Shepherd joins me in that, for the very interesting comments on our paper.

The subject is tremendous. My time is limited. I am going to answer Dr. Ferguson's question first. A great deal, I believe, depends upon the time of day at which the pressure is taken. We have found that if pressures are taken in the morning they are apt to be decidedly lower than if they are taken in the late afternoon or early evening, after one has passed through the activities of the day. Undoubtedly, a man can prepare himself for a blood pressure reading by depletion, diet, sedatives or nitrites before the pressure, if he knows he is going to have it taken again. Perhaps most applicants do not attempt any such procedure.

Mr. Shepherd referred to the possibility of carrying our investigation further. I hope we shall, not only with regard to heredity but also selectivity, when we have more cases, so that we can get more information with regard to not only the diastolic pressure but, also, albumin, casts, glycosuria, etc. Our paper necessarily had to be abbreviated because we had not enough cards to get sufficient information on the diastolic. I feel pretty certain that men who are running diastolics of 70 or 80 will not have as high a mortality as those running diastolics of 100 or more, and that the probability of longevity is far greater with a high systolic but a normal diastolic.

I think, if we consider the intricate nerve supply of the heart, if we have a normal organ and some sudden emotion, it is quite possible, in fact probable, that that heart, acting more powerfully under the stimulus of that emotion, will show a high systolic occasionally. Unfortunately, we cannot tell how frequently the occasions occur, but I think we can have some guide to it by the height of the diastolic.

One gentleman told me this morning that he did not think any case over 140 systolic was a standard risk. I am in hearty sympathy with that, provided the diastolic is low, but if the diastolic is up around, I will say, over 90, then it is a case for serious consideration.

There is one point that I have not heard mentioned this morning and that is the presence of faint traces of albumin or very few casts in the urine. I have felt for some time that, if we have an increase in blood pressure even though it may be very slight, and we also have traces, even 5 mgs. per hundred c.c., etc., we are playing with fire. We do not know whether or not the individual has had a glomerulo-nephritis in the past, and that his hypertension is not an essential hypertension but an actual one. We have to make a distinction between the two.

Gentlemen, if any of you have not read Dr. A. M. Fishberg's "Hypertension and Nephritis," I suggest you read it. It is authoritative and an extremely valuable contribution to the subject.

I am very sorry that we did not have more cases on which to base more definite conclusions, but we certainly hope to have them before very long and to give you something even more definite and something that I feel pretty confident will confirm, particularly in view of the statements that Dr. Livingston and Dr. Bolt have made, what we have already said.

We have one difficulty always in connection with our blood pressure readings and that is that so many examiners take their blood pressures three times, if they find the readings at all high, and then give us the third reading. In other words, we do not know what the high reading was. That is something we have got to overcome, and I think that when we get one reading and there is a suspicion in the case that the reading may have been higher, we should go back and get three more. I believe that is a very important point.

I am going to stop. I wish very much for the good of all the companies represented here that we might establish tables as quickly as possible, based on the fourth point. It is infinitely better than the fifth because I have seen differences between the fourth and fifth points of 35 millimeters and that makes a lot of difference when estimating the blood pressure and getting ready for a mortality investigation. It is true that the average is something like 5 or 6 millimeters between the two points, but it would not take a great many cases of the other kind to impair the value

of mortality statistics when they are based on the fifth point. I am thoroughly convinced that the fourth and not the fifth point is the point at which to take the diastolic and that it will be to the advantage of all if we take it at that point.

Someone has suggested here this morning, I do not remember who, that if we have a history of hypertension in the family, we should give it very careful consideration. Gentlemen, let me suggest to you that it is not a question altogether of a family history of hypertension but it is a question of degenerative change in the arterial system in the family, be that hypertrophy, hypertension, arterial changes, or whatever it may be, because they are all so intimately linked together that it seems to me we cannot exclude any one of these degenerative changes.

* * * * * *

Dr. Brown—Dr. Peter G. Denker, of the Equitable Life Assurance Society, has prepared for us a paper entitled, "Prognosis and Life Expectancy in the Psychoneuroses". Dr. Denker, will you present your paper?

PROGNOSIS AND LIFE EXPECTANCY IN THE PSYCHONEUROSES

A STUDY OF 1,000 DISABILITY INSURANCE CLAIMS

PETER G. DENKER, M. D.

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INTRODUCTION

It is probably true that "psychoneurosis" is the most frequently made diagnosis in medical practice. Culpen & Smith[1], in a routine examination, found that about 50% of factory workers, and 70% of students had neurotic symptoms, 16-20% of these symptoms being of fairly severe degree. Cassidy[2], in his consulting cardiological practice, notes that 29½% of persons seen by him are neurotic and show no evidence of organic heart disease, and this estimate is even exceeded by Houston[3], who believes that at least half of these patients presenting themselves as suffering from heart disease are cardiac neurotics. Of 2,000 consecutive ambulatory cases in the Boston dispensaries, 45% were psychoneurotic[4], and Lockhart, quoted by Gillespie[5], would have us believe that "as much as 60% of the time lost through sickness in certain industries is lost through psychoneurotic illness of some kind or another." The neurosis is not only frequent but is often prolonged in its course. Thus, Halliday[6], as Regional Medical Officer for Scotland under the Insurance Acts, found that one-third of 1,000 patients referred to him due to prolonged incapacity, and supposed to be suffering from organic disease, were in fact ill from neurosis. All these patients were rechecked by specialists and an independent observer. Similarly, Hoare[7], in his analysis of the disability claims of the Penn Mutual Life Insurance Company found that next to diseases of the heart, the most frequent cause of disability was disorder of the nervous system, constituting 16% of their claimants. It is interesting to note that the claim of longest duration (12 years) in his series was due to a neurosis resulting from

a fracture of the skull. Furthermore, in an analysis made according to duration of illness, the cases due to nervous system disease were found to constitute 40% of the total number of claims, in this respect leading the field by a large margin. The economic loss to the individual and to industry due to this impairment is, therefore, obviously severe.

Outline of Study

It was thought that a follow-up study of 1,000 consecutive disability claims due to psychoneurosis might add some enlightenment to this complex problem. These claimants were all insured in the Equitable Life Assurance Society, and if totally disabled received varying amounts of money in monthly payments depending on the amount of insurance carried. Most of these policies were issued between the years 1915 and 1930, the inception of their disability occurring from one to twenty years after issue. The diagnosis in all instances was made by a physician and often corroborated by a nerve specialist or psychiatric hospital, and in the New York area most of the cases were personally examined by the writer. Total disability was only recognized after the insured had been ill for at least three months and to an extent that would prevent him from engaging in any occupation for remuneration or profit. The milder or more transient neurotic disturbances are, therefore, automatically excluded from this study. Lastly, the follow-up study was made at least five years, and in many cases 10 to 15 years after disability had commenced, results being obtained by correspondence with the insured or his physician, examination by the Society's examiners, personal interviews or inspection reports. Due to the cooperation of these various informants, 100% of the cases were traced and brought up to date.

Great Frequency of Error in Diagnosis

The great risk of error in too glibly labelling a patient a neurotic was apparent early in the study. On following the cases it was found that 293, or 29.3% of the total 1,000 cases had been erroneously diagnosed as "psychoneurosis", the true nature of the

illness manifesting itself in most cases within one year, as some organic disorder. There were 62 deaths in this group of 293 cases, an unusually high percentage. It is interesting to note that in a follow-up study of 100 patients diagnosed as "neurosis", Comroe[8] found that 24% showed definite evidence of organic disease within an average period of eight months, never after two years, figures closely approximating ours. In most instances rechecking of the original hospital records of these patients revealed that symptoms suggesting the organic disease were present during the original admission. He likewise experienced a high mortality rate, 7 of the 24 cases dying within two years. A tabulation of these 293 cases erroneously diagnosed neurosis follows:

TABLE I

END RESULTS OF CASES ERRONEOUSLY DIAGNOSED NEUROSIS

1.	Psychosis (Manic Depressive, Schizophrenia, involutional melancholia, general paresis, etc. (suicides —20)	149
2.	Cerebral arteriosclerosis and hemorrhage	21
3.	Other Organic Brain Disease	25
4.	Pulmonary Tbc. or Lung Abscess	17
5.	Organic cardiac disease	13
6.	Hypertension and nephritis	14
7.	Cancers	7
8.	Cholecystitis or Cirrhosis of Liver	8
9.	Duodenal Ulcer	4
10.	Chronic Arthritis	7
11.	Miscellaneous	28
		293 Cases.

It cannot, therefore, be too frequently emphasized that the pitfalls in diagnosing a "neurosis" are deep and that the diagnosis should only be made after a most meticulous exclusion of organic disease has been carried out. It would probably be wise not to insure an individual for at least 6 months to one year after recovery from a neurosis.

It might be appropriate at this point to mention the extreme difficulty which is occasionally encountered in differentiating between the neurotic and the malingerer. The malingerer realizes he is not ill and consciously simulates his symptoms, whereas, the

neurotic is genuinely convinced that he is sick and is usually unaware of the emotional factors related to his difficulties or the purpose that his symptoms are serving. The inconsistencies in history and examination, as well as the frequent exaggeration of symptoms or signs by the malingerer help in diagnosis. To make matters more difficult the two conditions may coexist. Foster Kennedy[9], has aptly described this state, "hysteria and malingering are often dovetailed and come together in one mind, and many symptoms begun as malingering end as hysteria by suggestion. In the great majority of cases, one cannot tell easily what is virtue and what is vice. Just as few of us know if we are all good or all evil, we are in truth neither black or white, just gray."

The remainder of this paper will concern itself with an analysis of the 707 true neurotics in this series.

Age

The age of onset encountered in the study varied widely from 19 to 59 years, the average age being 37.2 years. This is in contrast to the slightly higher age average of the erroneously diagnosed group in which 40.8 years was obtained. The chances of error in diagnosis is, therefore, somewhat greater at the higher ages.

TABLE II

AGE AT ONSET OF DISABILITY

Age at Onset	Male	Female	Both Sexes
–19	2	....	2
20–24	17	38	55
25–29	43	61	104
30–34	64	82	146
35–39	67	55	122
40–44	75	33	108
45–49	57	30	87
50–54	39	19	58
55–59	20	5	25
Total	384	323	707

It would seem that middle age in the male produces the greatest neuroses, possibly because his responsibilities are greatest and his career made or broken at this period. In the female this age inci-

dence is somewhat lower. As Halliday[6], puts it, "he tends to look before and after and pine for what is not, and by means of an illness escapes the responsibilities of an immediate or future environment hard to contemplate steadily." Burns expressed the mood when he wrote,

"But, oh, I backward cast my ee on prospects drear;
An' forward tho' I canna see, I guess and fear."

Sex

Of the 707 cases, 384 were males and 323 females. 45% of these claimants were, therefore, females, as contrasted to the fact that only 12-14% of the insurance policies during these years were held by women. Neuroses were, therefore, 3 to 4 times as frequent among female as male insureds, which corresponds with the clinical frequency of female neurotics. Of 100 neurotics studied by Bennet & Semrad[10], 73% were females and 27% males, and 61% of Luff and Garrod's[11] series were females.

Occupation

Occupationally claimants might be classified roughly as follows:

TABLE III

OCCUPATION OF DISABILITY CLAIMANTS

Clerical workers	185
Executives	170
Teachers	132
Merchants	88
Professional workers	54
Farmers	33
Nurses	23
Students	13
Manual workers	7
Housewives	2
	707

No definite conclusions can be drawn, but the high incidence among clerical workers, executives, teachers and professional people contrasts forcefully with the greatly diminished number of

manual workers and housewives. On looking over the case histories, one was often struck by the monotonous existence of the occupational misfit, the clash of temperament and intelligence, etc. "Blessed is he who has found his work; let him ask no other blessedness," wrote Carlyle, and Huddleson[12] has worded the problem well, "whatever his personal and family histories have been, any individual may find himself in a situation that tends to become progressively more unbearable and so predisposes to psychoneurotic conduct. A man working at tasks beyond his intellectual or physical capacity, or his emotional endurance, and harrassed by obligations to carry on exhausting work without being able, from whatever ultimate maladjustments, to derive satisfaction from his work, this man is becoming predisposed toward a neurosis. Bare failure is intolerable; but failure camouflaged and compensated for by an illness that brings attention, sympathy, rest, and often an indemnity or wages without work, is far from intolerable."

Family or Past History of Mental Disease

Though a history of familial mental taint is frequently obtained in the private neurotic patient, such a history was only rarely given in this series. Thus, there were only 35 individuals of the 707 who presented a history of nervous or mental disease in either themselves or their family, an incidence of about 5%. Yaskin[13] in a review of 100 psychoneurotics obtained a positive family history of neuropathy in 60%. This discrepancy is probably explained by the absence of a special question on the insurance application as to mental disease in the family, as well as the fact that the average insurance examiner is not neurologically minded. Lastly, lack of candor on the part of the applicant plays an important role.

Life Expectancy

A thorough search of the literature revealed no accurate mortality study of people who have suffered from a neurosis. The following table expresses our experience in this series of 707 cases:

TABLE IV

MORTALITY STUDY OF NEUROSES

Attained Age	Deaths	Males Expected by U. S. Table	Males Expected by E. L. A. S. Table	Mortality Ratios U. S. Table	Mortality Ratios E. L. A. S. Table
–39	4	5.36	3.42	75%	117%
40–49	5	9.62	7.13	52	70
50–59	8	12.08	10.96	57	62
60+	1	3.70	3.65		
	18	30.76	25.16	59%	72%
		Females			
–39	..	6.24	4.62		
40–49	2	4.48	4.28		
50–59	1	3.85	4.62		
60+	1	.71	.87		
	4	15.28	14.39	26%	28%

This study was made from the inception of the disability to the date as of which we were last able to trace them. There was a minimum of at least 5 exposure years for each case, with a total of 2,981 exposure years for the 384 male cases and 2,526 years for the 323 female lives. The average number of years of follow-up was, therefore, 7.6 years. The expected deaths were calculated by 3 mortality tables: (1) A table representing the Society's own experience between 1920 and 1932 on standard insurance policies in force more than 10 years (this was applied to both the male and female experience), (2) the U. S. White Males Table representing the experience during the period 1928-31 of the total white male population of the continental United States, and, (3) the U. S. White Females Table corresponding to the Males table. Each of these two tables was applied to the corresponding sex in this present study.

There was a total of 18 male and 4 female deaths in the entire series, death being due to various causes. The expected number of male deaths was 30.76 deaths according to the U. S. Table and 25.16 deaths in the Equitable Life Assurance experience, giving a favorable mortality ratio for the entire male group of 59% and 72% respectively. In the female group, only 4 deaths were experienced giving a mortality ratio of 27%. It is, therefore, evident that psychoneurotics have a life expectancy which is better

than normal. This is probably due to the excessive care and pampering they usually undergo, their escape from the winter rigors to southern climates, long vacations, frequent rest periods, etc. There would thus seem to be statistical proof for the expression that neurotics are more likely to wear out their families and friends rather than themselves.

A word as to the frequency of suicide among neurotics. Though the fear of committing suicide is a frequent symptom in patients suffering from anxiety states, and hysterics often threaten to do so, the risk of a person committing suicide who is suffering from a true neurosis is very small, and the old saying that these patients are too fond of themselves for this to be a serious risk is a true one. In the 22 deaths in this series there were 3 suicides. Jamieson[14] in a study of 100 suicidal deaths found only four in psychoneurotics; these were in the acute phase with morbid fears, depression and various hypochondriacal symptoms resembling a mixed manic-depressive reaction. It is interesting to note that over two-thirds of his 100 cases occurred in the course of a psychosis of the manic depressive type. T. A. Ross[15], in an experience of over 1,000 cases of neurosis encountered only 7 suicides; once again the accuracy of diagnosis must, therefore, be stressed. The psychoneurotic should be carefully differentiated from the psychotic. The small suicide hazard in the neurotic is in marked contrast to the much greater risk of this tragedy in the psychotic. Strecker and Palmer[16] have clearly differentiated the two, "a psychoneurosis is essentially different from a psychosis. The psychotic is profoundly shaken in his whole personality and sometimes disintegrated; there is a deep cleft between self and environment, and reality for him is disturbed, distorted and even abolished. In the neurotic there is only a partial personality altering and environmental contacts remain real and relatively undisturbed. Meyer sees the psychoneurosis as part reactions, half way places between normal and psychotic, maladaptions to be sure, but nevertheless, compromises that recognize and concede the claims of reality. Finally from the standpoint of insight, the capacity to 'stand off' and 'look in', survey and judge, even if not understand, here the psychoneurotic overwhelmingly demonstrates his freedom from the psychosis."

The following general classification of the two conditions is appended for purposes of clarification:

1) Psychoneurosis: types,
   a) Anxiety
   b) Hysteria
   c) Neurasthenia or Psychasthenia
   d) Compulsive—obsessive
2) Psychosis: types,
   a) Manic-Depressive; involutional melancholia
   b) Schizophrenia (dementia præcox)
   c) Organic types (paresis, brain tumor, encephalitis, epilepsy, etc.)
   d) Toxic (bromides, luminal, hasheesh)
   e) Alcoholic

We encountered no case of neurosis later developing into a psychosis, an experience likewise common in practice and the medical literature.

Duration of Neurosis

Of the total of 707 cases,

45.4% terminated within 1 year of onset
27.2 " during 2nd year
9.1 " " 3rd "
4.3 " " 4th "
3.7 " " 5th "
10.3 were still disabled 5 years after onset.

Approximately three-fourths of all cases, therefore, recover within two years of the onset of disability and a little less than half within the first year. In the follow-up series of Luff and Garrod[11], three years after the patients had been discharged from the clinic, 55% were found to be in good health, the results being more or less identical for all therapeutic methods used, including analysis. In our series, 20 cases had been treated by lengthy analysis with approximately the same degree of success as the remainder of the cases treated according to various other procedures, making one doubt the peculiar therapeutic efficacy of this procedure. Yaskin[13] in his series of 100 neurotics found that 41 recovered, 41 improved and 18 had not been benefited by treatment in hospital or clinic. He like-

wise felt that for most of the patients suggestion, encouragement, medication and partial analysis of the precipitating conflict was sufficient.

It was thought interesting to attempt to ascertain if any relationship existed between the amount of disability income and the duration of the illness. The following table shows the persistency of these claims over a five year period from the initial date of the disability, expressed on the basis of percentage of the total claims in each category.

TABLE V

MONTHLY DISABILITY INCOME GROUP

Duration of Disability	All Claims	Waiver Only	Less than $50	$50–$99	$100–$199	$200
Less than 1 year	45.4%	31.6%	47.4%	43.4%	45.8%	41.9%
1 year or more	54.6	68.4	52.6	56.6	54.2	58.1
2 years or more	27.4	36.8	25.0	30.2	23.6	35.5
3 years or more	18.3	26.3	16.8	18.7	16.7	25.8
4 years or more	14.0	15.8	12.2	14.8	12.5	24.2
5 years or more	10.3	15.8	8.2	11.0	8.3	22.6

It can readily be seen that in the higher disability income brackets the duration of the claim is likely to be longer. Thus, whereas only 10.3% of all claims lasted 5 years or more, in the disability income group receiving $200 or more per month, 22.5% of the cases lasted this length of time. Furthermore, it would seem that after the third year in this higher income group the percentage remains almost constant, so that it is fair to say that neurotics receiving $200 or more per month, who have been ill for three years, practically never get well. It would seem that the monthly disability check eliminates the incentive to get out and fight life's struggles, and in addition, it acts as a constant reminder to the highly suggestible, mentally-ill patient that he is disabled, and an invalid, and recognized as such by the outside world. Instead of helping to rehabilitate the neurotic, we are doing the worst thing possible for his mental health; constantly impressing upon him that Society looks upon him as useless and unable to carry on. How much wiser we would be, to pay these claims by some form of lump-sum settlement at the end of the first year of disability or sooner.

Lump Sum Settlements

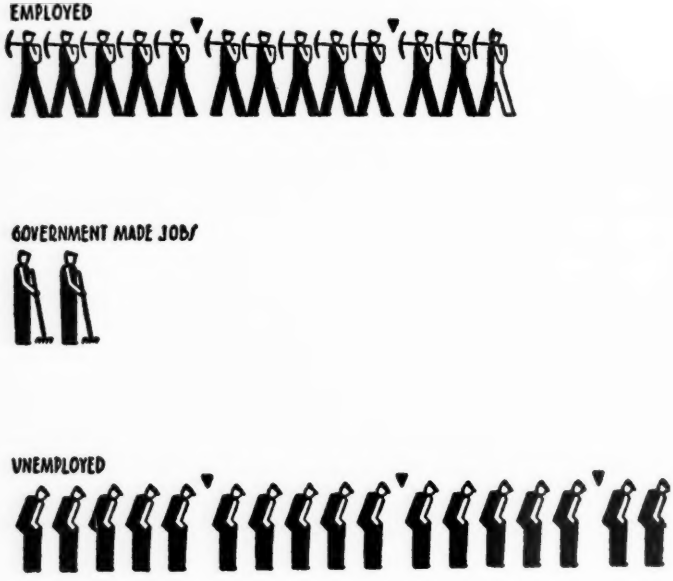
In the last few years, much has been written pro and con about "lump-sum settlements". In Denmark, compensation is paid in a lump-sum at an early stage of the incapacity and 93.6% of the injured recover from what in this country would be called a traumatic neurosis. On the other hand, the sick man in Germany is entitled to a monthly pension and only 9.3% recover from the same disease[17]. Jolly[18], has had a similar experience with industrial hysterics, finding the percentage of cures high when lump-sum settlement is speedily made, whereas it is low where the individual draws weekly compensation. Foster Kennedy[9], quotes Dr. Lewy, who is chief medical examiner for the compensation commission of the State of New York, as saying, that he has "never known of a single case from my very large material, which was ever disposed of, unless the individual received a monetary remuneration to his own satisfaction." Norcross[19], on the other hand, in a follow-up study of compromise agreements, settled by lump-sum in New York State, found the system used did not produce successful results, in that, in most cases, the claimant's physical health or mental attitude was not improved, nor did it stimulate him to find work or aid in the conservative expenditure of his compensation money. Marked improvement resulted from the settlement in only 17 of 64 cases. He attributes most of the failures to unnecessary delay in the compensation rooms, extended litigation and unwise handling of these cases by the insurance carrier or their representatives. "The group which understands this type of man best, namely the State Compensation Medical Staff, often makes recommendations which are not followed." He recommends that the interval between the onset of neurosis and the settlement should be shortened, investigations should be speeded up, and neurotic claimants should not be given copies of their medical reports or hear medical testimony which might cause them further anxiety or worry. They should not be advised that it is legally possible to reopen their cases at a later date if their conditions change for the worse, since such knowledge is harmful to the neurotic patient. "Work of any kind is the best therapy", and better cooperation

from the insurance companies would return more claimants to work much sooner.

No.8

EMPLOYMENT STATUS FOLLOWING SETTLEMENT

Each figure represents 10 men



from Norcross, Carl—
Vocational Rehabilitation & Workmen's Compensation—
Rehabilitation Clinic, N. Y. 1936.

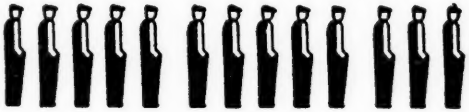
No.11

HEALTH OF NEVROTIC MEN FOLLOWING /ETTLEMENT

Each figure represents one man

EXCELLENT

GOOD



FAIR

POOR

VERY POOR

DEAD

from Norcross, Carl—
Vocational Rehabilitation & Workmen's Compensation—
Rehabilitation Clinic, N. Y. 1936.

The medical literature is full of warnings about the futility of closing neurotic cases a long time after the accident. Though writers differ as to the manner in which claims should be settled, all agree they should be settled promptly and be rather small, and that prolonged litigation and appeals are the worst things that can happen to a psychoneurotic. All the emphasis is on *early* closure. Huddleson[12] writes, "If the final settlement is delayed for a number of months, the neurosis is not immediately alleviated by the indemnity and may become quite unaffected." Sir John Collie[20] refers to the protracted legal delays relevantly by saying "In cases which are contested, the 'Law's Delay' accounts for a great deal of prolonged disability, and the development meantime of functional disease. The close questioning, oft repeated, as to the exact details of the accident and his feelings, by both lawyers and doctors, and the everlasting inquiries of well meaning but injudicious friends and acquaintances, all help to rivet the man's attentions on his internal anatomy. Too often his abnormal mental condition will remain until the dispute arising from the accident is settled, and generally, so far as recovery is concerned, it matters less than one would expect whether the claimant wins or loses." An educational campaign whereby the legal profession, insurance adjustors, and legislative arbitrators and physicians could grasp the conception of neurosis, and learn to realize the advantages the neurotic obtains by being ill would go a long way toward intelligent handling of these cases.

In this series, 15 claims were settled by compromise and lump-sum payment; usually this included the repurchase of the disability clause by the Society. In five of the fifteen cases, no clear-cut opinion could be rendered as to whether or not benefit had been obtained by this form of procedure. Seven cases, however, were followed by distinctly favorable results, the patients recovering rapidly and remaining in good health for a long period of years. In the three remaining cases, no beneficial effect was obtained, the disabling neurotic symptoms persisting for long periods following settlement. An attempt was made to analyze the reasons for the success in some cases and failure in others, and the following table

lists the results in those cases where a definite opinion could be offered:

TABLE VI

Cases Successfully Terminated by Lump-Sum

	Age at Onset of Disability	Duration of Disability Before Settlement	Amount of Disability Income Received
1.	28	5 years	$ 50.00 month
2.	33	1 year	100.00 "
3.	29	2 years	500.00 "
4.	31	1½ years	50.00 "
5.	33	1½ years	400.00 "
6.	43	1½ years	840.00 "
7.	37	6 months	250.00 "

Cases Unsuccessfully Terminated by Lump-Sum

	Age at Onset of Disability	Duration of Disability Before Settlement	Amount of Disability Income Received
1.	44	3 years	$100.00 month
2.	48	4½ years	65.00 "
3.	42	3 years	250.00 "

It is clearly seen from the above tabulation, that (1) cases in the younger age groups (roughly under 40) are more favorable for lump-sum settlements. (2) The shorter the duration of disability before settlement is made, the better the outlook of success. It would also seem from the above that the amount of disability income is not as important a factor as are the two previously mentioned. A claimant receiving $840 a month, who had been ill for one and one-half years, was promptly cleared-up of mental symptoms by settlement of the case in one sum, and was soon back at work functioning normally. Experiences of this type make one feel that the situation with the disabled neurotic is far from hopeless, and that intelligent handling of this form of disability could go a great way toward minimizing the severity of the problem.

Summary

1. A follow-up study of 1,000 consecutive disability claims for psychoneurosis was made, at least five years after disability had commenced.

2. Almost 30% were found to have been incorrectly diagnosed, the true nature of the illness manifesting itself within one year.
3. A mortality study of the series shows that neurotics have a distinctly greater life expectancy than normal. The rarity of suicide in this group is stressed.
4. The duration of neurosis in relation to disability income, type of therapy, age, sex, and other factors is discussed.
5. The cases settled by lump-sum compromise are analyzed, and this method of terminating neurotic claims is reviewed in the light of our experience as well as others.

NOTE—I am greatly indebted to Mr. John Mills, Assistant Superintendent, Bureau of Statistics, Equitable Life Assurance Society for many valuable suggestions and statistical analyses in reference to the problem studied.

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DR. BROWN—From Gowers and the great school of English Neurology at the Queen's Square Hospital at London there came to America, just at the turn from the first to the second decade of this century, a young man to work with Dr. Charles Dana of

Cornell. And he it is who is to discuss the paper which is before us. He was the first Chief of the Neurological Service in Bellevue. He is Professor of Clinical Neurology at Cornell.

It is said that his writings in the field of Psychiatry and Neurology are too numerous to mention in the few hours which we devote to these meetings. Psychoneurosis and its relation to Industrial Compensation is a favorite subject with him. Because of his large experience, he has been of great consultant value to insurance companies. It is, therefore, a real privilege and pleasure for me to introduce Dr. Foster Kennedy.

Dr. Kennedy—I feel rather like a whited sepulchre, full of dead men's bones, because last night I made the worst diagnostic error of my life in that I told Dr. Brown that a waiter who was manhandling him was apparently drunk. I have a very distinct drop in my positive self-feeling this morning in consequence and I am not sure that my morale is not permanently lowered.

I think we live not just by bread alone, but also by catch words. The term "traumatic neurosis" is one which, by the conjunction of adjective and noun, seems to prejudge that the neurosis is caused by the trauma. I believe it is a term which should not be used. It is permissible to say neurosis following accident and allow the sequence to be unjudged. Further, I do not believe in the existence of a "traumatic psychosis," which is in the literature of nearly every medical hospital.

There is such a thing as a traumatic psychosis which immediately follows brain laceration but a "traumatic" psychosis of functional type, I do not believe exists.

We hear the word "suggestion" bandied about in the lay press and in medical literature. What is suggestion? I would define it as the acceptance of an idea which is in consonance with an already established emotional trend, whereas "criticism" is the resistance to an idea which is out of consonance with an already established emotional trend. Everybody is suggestible. We accept a great deal of our information by straight suggestion. Most of us buy a Republican newspaper if we are Republican because it elevates our sense of ourselves in that we find our own views in the paper

and we agree with our own views, particularly when we see them printed.

We would go to the stake on behalf of a classical education if we have been exposed to one.

We accept the idea that the moon is responsible for the tides, and which of us knows anything about it? It has not really been proven. It is a hypothesis which seems to fit the facts and we would die on behalf of it.

We are all suggestible. Suggestion is like hitting a baseball the way it is going, on the tail, whereas criticism is like hitting it the way it is coming, on the nose.

If you give a man an idea which is in consonance with his own wishes, conscious or unconscious, that idea becomes part of his personality and is built into his organism. Therefore, every word that a doctor utters in the presence of a patient, after an accident involving compensation, is a suggestion of enormous emotional power.

The volume of cases awaiting compensation in this country is estimated as about one-sixth of all the hospital admissions and Peter Denker has just found that in a thousand consecutive cases there are 30 per cent. errors in diagnosis. That is a terrible figure. It is a figure which is highly dangerous because an error in this field is a very important error when we find that inside of two years of that mistaken diagnosis patients to the number of 25 per cent. died.

Why do we make those errors? It is for lack of accurate technical knowledge. The only possible way in which you can make a diagnosis of a neurosis is by knowing organic conditions so thoroughly that as soon as you see the half-appearance of one you know it for what it is; that it is an unharmonious picture, that it is not a picture in harmony with a necessary organic state, necessary to be an organic state.

Therefore, accurate technical knowledge must be exercised to sift the wheat from the chaff, to sift organic from inorganic, to sift structural from what might be called psychological. Furthermore, exact technical knowledge is the only possible instrument possessed by the doctor for curing these people.

The quack cures in virtue of his ignorance; the doctor must cure in virtue of his knowledge. The ignorance of the quack is his forte because his own confidence to cure is not impaired by his knowledge. He tells everybody, "You will get well".

The doctor, exposed in a small way to pathology, remembers at the bedside of a paralyzed patient the look of a spinal cord bearing marks of degeneration, then he may *feel* that that individual is suffering from hysterical paraplegia but if he be not absolutely certain, he cannot cure the patient.

I watched Coué going from patient to patient saying exactly the same thing to all kinds of patients. His was the reverse of the scientific method. He knew only his successes; he was blind to his failures, so his confidence kept on being piled up within himself.

I remember in France a very good medical officer asking me to come and see a man with a wound in the arm. As we went to the hut, this medical officer said, "I think this man is hysterical because I don't see how he could be paralyzed in the way he is from his wound".

On examination, the man had a small through-and-through puncture of the deltoid and a completely paralyzed or static hand, very recently acquired, which could not have been caused by the wound. I was able to tell the man, having made up my mind about it, that he must concentrate on trying to move one finger. We worked together and he had a little, almost involuntary twitch, which surprised the man very much, and then with moral suasion and pressure, he began to move his hand and was cured.

As we came out of the hut, the medical officer said to me, "Why couldn't I do that? I knew that man was hysterical". I said, "I'm damned if you *knew* he was hysterical! You only thought he might be".

It is the subjunctive mood that kills the thing. You cannot cure those people unless you have a categorical imperative inside yourself and that can be produced only by a sense of knowledge within us because we know a wider range than the quack. We are not born with that confidence that the quack has just by nature. In fact, most of our experience and most of our training tend

to make us doubtful, careful, to look over a wide range, to regard all possibilities. The quack has not that disadvantage, and unless we go along the path in which we are educated sufficiently far to feel our own power, then we can never communicate power to others. We must have exact technical knowledge in this domain if we would diagnose and cure disease-pictures assumed consciously or unconsciously.

I think one can define hysteria as an unconsciously assumed picture of disease, whereas malingering is a consciously assumed picture of disease with a clear objective and aim on the part of the assumer.

What does one mean by an assumed, or an unconscious assumption of a disease-picture? That brings us back to "suggestion". If you tell a man that he has paralyzed the nerves of his arm and he believes you, he will lose the ability to throw the switches of his will into the innervating centers of that arm. He may do it with no sense of conscious aim. A man may believe an error in diagnosis so completely that it becomes for him a fact.

In February of 1918, for instance, I was assigned temporarily—having just come out of hospital myself—to King George's Hospital for light duty. There I saw some of the older and longer-standing cases of the war, not only medical cases but psychotic and neurotic cases.

There was referred to me for an opinion by a surgeon a demobilized soldier who had been a gunner. The situation was this: This gunner, in May of 1915, two and a half years before, had received a large superficial wound in the inner side of the left arm, the arm had been put up at right angles for drainage for six weeks; it had healed, but when taken down, it was found to be completely paralyzed, atrophied and withered. He was put before many boards of "brass hats" and he was certified as having a complete paralysis of the brachial plexus. He was discharged from the army. He got his silver badge to wear on his civilian clothes, "For King and Country, Services Rendered". He was compensated for the complete loss of his left arm. He was given that bonus. He got a job as an elevator boy through the Red Cross, which he could do with his right arm. Altogether, he

thought it was a fine war; he thought it was a very good war, and he had no kick at all.

He was allowed, as were all discharged soldiers, to come to military hospital for anything he wanted, and he turned up in February, 1918, with a demand that the arm be taken off at the shoulder. He was accustomed to seeing all his friends get their arms taken off and their legs taken off, and as he put it to the surgeon, he was fed up with putting this useless arm into his clothes every morning. The thing was in his way and he thought life would be much simpler if he just had the useless thing lopped off.

The surgeon sent for me, before he did that, to know if it would be worth while to do the very complicated task of dissecting the brachial plexus out of the scar, and asked if by so doing there was any chance of restoring function. I remember that the man was sitting in his trousers when I examined him. He was stripped to the waist.

(Just as an aside, I am sure that nearly all medical mistakes are made by not taking people's clothes off. I am sure that must be laid down to medical students. We must keep telling ourselves that examining people through shirt-windows is pure folly. I remember a man whom Philip Kerrison sent me because he was getting *shortsighted* and deaf at the age of 56. I stripped him and went all over him and still could not see any reason for it. Kerrison thought he might have a cerebral tumor. He was dizzy and unsteady on his feet. I noticed that he had a wig on and bade him take it off; the diagnosis lay under that wig. He was a case of Paget's disease. I could not discern it as long as he had that wig on. His Paget's disease produced enlargement and deformity of the head and had so changed the focal length of the eyeball that he had become very shortsighted and almost blind. That was the reason of the shortsightedness in his later years.)

But to go back to the soldier: The man was sitting undressed in the chair and I told him to shrug his shoulders. He shrugged one shoulder. He lifted up the good shoulder. It struck me that he must have ankylosis in some way from prolonged immobilization; he could not use his trapezius muscle, but he certainly could

not have a paralysis of the trapezius muscle from an injury to the brachial plexus of the arm.

To cut a long story short, he was a case of hysteria. He had a minor organic condition; a partial paralysis of the left ulnar nerve but only a partial organic palsy. The rest of the condition was in the utter acceptance by him of the idea of total palsy. So firmly did he believe the mistaken diagnosis that he wanted to get the arm off though by doing so he had absolutely nothing to gain; he was no more remote from the war by an amputated arm than he was with a certified arm, no more.

And so, hysteria is a very real thing. It is the utter, uncritical acceptance of an opinion. The opinion, in order to be completely, "organically" accepted, has got to be in consonance with the emotional trend. Of course, everybody wants to be an elevator boy rather than be killed in the front line. By 1918 nobody longed for the front line unless they were very foolish.

The same situation operates in the civilian in peace time if he be injured or he has had some sort of accident. There follows then what Charcot used to call the "period of meditation", about three days, during which time perhaps a gloomy diagnosis is made by his doctor, rapacious relatives discuss the possibilities of what might happen to him, and a lawyer appears;—a very short period of that kind of suggestion and optimistic acquisitive thinking is enough to produce a large sequence of symptoms. It is then that it is necessary for the state and for insurance companies and for the doctor who wants to restore a good workman to his own happiness to make an accurate, technical diagnosis. That is where you need the technical knowledge to prevent the occurrence of neurosis. If you know what is the matter for certain, then you can stop the development of subsequent improper ideas.

People often tell me that there must be something wrong with such and such a man because he can make $50 a week and gets only $18 by way of compensation. The answer to that is the experience that we have had in Federal relief in this country, in unemployment insurance in Great Britain. It is a confession of the failure of our civilization to make the work of the average working man sufficiently interesting to him so that the average man, and

those of the weaker fabric particularly, prefers secure idleness to insecure struggle. If we get, as Peter Denker said, security on however small a living, we prefer or think we prefer to be idle rather than to be busy. The man who believes that is on the whole wrong; he is happier lacking the necessities of invalidism, but he is unaware of this because he does not truly enjoy his work and so prefers to live idle on $18 than to work for $50.

In addition, too, we must remember the validity of his symptoms if he has had them long enough; how they become true for him. I always say that the colonel who first tells his fish story knows, if you backed him up into a corner with a gun, that the fish was only *so* big although he said it was so —big. He knows the size of the original fish and has insight under compulsion into the fact that he is a liar. He might be said to be a malingerer. But after thirty years of that fish, and the fish has gotten to be *that* big, he is not a malinger any more; he is a hysteric because he is no longer conscious that he is a liar. He has accepted the truth of his own fish story. And that happens to all of us.

I wonder if you would permit me to interrupt myself and the trend of this talk to say a word about head injuries which have a peculiar importance in your work.

A man injured on "the outposts of Empire", in his big toe or his arm or his thigh, has not as large a psychical, emotional superstructure as if he had a blow on the head. He feels that he had been hurt pretty near where he lives if he has been hit on the head, and furthermore, we all have a certain amount of mystical apprehension about what is inside either our own head or the head of another person; I mean, we feel that something queer might have happened to the brain, which we feel reasonably certain has not happened to the liver, in taking a blow over the casement of one organ or the other.

Consequently, we ourselves, we doctors, are inclined to say that strange symptoms which we cannot understand, following a head injury, might be due to some high-sounding word like commotio cerebri or "multiple petechial hemorrhages". One hears this as a very common pathological speculation.

I have been struck by the very different prognoses in head injuries in footballers or polo players and head injuries sustained by workmen. They seem to follow a completely different course.

A polo player and steeplechase rider, a gentleman jockey, whom I took care of, was knocked out by a fall and had a fractured skull, bloody spinal fluid, double Babinski sign, and was disoriented for two or three days. He woke up and said he felt fine and wanted to get up; he was much annoyed because he was kept in bed five or six weeks. Then he became entirely well with not a word of complaint of subjective symptoms.

A patient who was sent down to Bellevue by the Mayor, a son of a friend of his, was another good example. This man owns and runs a restaurant. In July he had a motor-car accident. He was taken into hospital, and I subsequently got the report from the hospital. He had been unconscious for three days, a very ominously long time, and was inaccessible for a week more. He was kept in the hospital for only a month altogether, (too short). He had had bloody spinal fluid. He was not kept in the hospital nearly long enough under the circumstances. I think he resumed work in the end of September.

He came to me, under family pressure, for an opinion about his condition. He had no complaints except a certain stiff feeling at the back of his neck. He had no headaches, he was never dizzy; he said he had some difficulty in remembering the names of patrons in the restaurant sometimes. He was more slow-minded than he liked.

Upon examination, that man had a left Babinski sign and had a brain laceration of very definite importance. He is going to get perfectly well although slowly, but his complaint is almost nil. He gets down to the market and does all the meat buying, which is a taxing occupation, and he pinch-hits for the head waiter when he is off duty; he meets all the patrons, and he is doing far too many things for a man in his condition, but the point is he has no complaint. Had he been wrangling through the compensation courts, he would have been, by now, a bowed old man after such an injury.

In order to try to find out what kind of a *skull* injury is requisite to produce a *brain* injury, Dr. Wortis and I investigated about a thousand cases of acute head injury, and we laid down certain criteria for what should constitute an acute head injury.

Without some such standards to guide him to a reasonable decision in this matter, a neurologist is like a captain sailing a sea without a chart. What measure of evidence must there be to warrant a conclusion that the head injury is sufficient to produce tissue changes in the brain? That is the point.

The following criteria are offered you for determining the degree of disability caused by organic neural injury; first, absolute criteria. One, X-ray evidence of skull fracture. Two, bloody spinal fluid. Three, bleeding or cerebro-spinal fluid leakage from orifices, especially from the ears. Four, focal cerebral paralysis or weakness.

These are four criteria which are absolute proof of neural injury after a skull injury.

Presumptive criteria: In the order of importance, convulsive states following injury, ventricular distortion following injury, history of prolonged unconsciousness, and last, history of adequate trauma, with special consideration of the occurrence of vomiting following the injury.

These are appreciable units and they are implements for appraising brain injury. We can absolutely and accurately determine the four units of absolute criteria and also the units of 7 and 8, in the second group, which are history of prolonged unconsciousness and history of adequate trauma.

A patient often complains of convulsive state and comes under observation. The inducing of an attack in doubtful instances by the use of a convulsive agent or hyperventilation offers a means to confirm or deny the truth of it.

We used to do that in France quite commonly. A man would have a fit in the front line and he would be sent down naturally very quickly, because they did not want an epileptic running around the trench with a gun. He would land in the C. C. S., not having fits but with the history of fits. They were fairly common and one had to be careful that they really did have fits. If one could not

get a very good, clear story, which one often could not, we had to give them cocaine. We gave them an injection of cocaine because that is a convulsant agent in any epileptic, and if then he failed to have a fit, he had to go back to the line.

Headache and dizziness following a head injury are unmeasurable and they have very little weight in establishing the presence of an organic lesion. If a man under sixty has a head injury, it is probably less important; but in a man over sixty it is quite a different matter than a head injury in a young man, or even a middle-aged man. Especially if there be a degree of arteriosclerosis, head injury is badly received. In prognosis, I would not be dogmatic about the man of sixty or over, after a head injury, but I would feel dogmatic about a man under sixty.

If a man under sixty years of age continues to complain of headache and dizziness after a period of four months, unassociated with any of the first conditions for the validity of brain or meningeal injury that I have put down, I feel that headache and dizziness are to be rejected as indications of structural change and are to be accepted as arising from a suggestion neurosis.

So much for the note on head injury, which I thought might be interesting to you.

I was very much interested in Peter Denker's statement about Denmark and Germany. In Denmark, which deals with cases of a lump settlement and quickly, they have a cure in 90 per cent. of the cases, and in Germany, with about the same type of human integrity as in Denmark, they can cure only 9 per cent. under the idea of a weekly or monthly stipend. I think that is the very crux of this question. These people cannot help but get ill and stay ill if they are supported by weekly payments and they are hailed interminably before industrial commissions or courts for many hearings.

I complained only yesterday to the Industrial Commissioner in Worth Street of the unmedical situation whereby a patient is allowed to sit in a court room and hear his lawyer claim that he is permanently and totally disabled. I said to the Commissioner, "If we would talk in that way in a sick room in a private house, we ought to be barred from our profession. We would make our

patient very ill if we were to sit by the bedside of Mr. MacSnipe in his private house and say, 'Of course, poor Mr. MacSnipe will never, never work again' ".

The idea of our doing this is so absurd that it is only possible to see the absurdity of our conduct with relation to the injured workman when the two procedures are put together, are placed in contrast.

Once we know that a patient has a neurosis that is the result of suggestion, of fixed ideas, we must quickly give him a compensation, whatever is necessary, and close the case beyond the possibility of its being reopened. That is where law makes it very difficult for medicine to act properly in this matter.

Before I stop, I will tell you of two or three cases of my own experience that might interest you. One was a Polish workman who was injured, a matter of fourteen years ago, in the head, this way: He was going down the Merrick Road at eleven o'clock at night in a closed motor car. There was a hole, unlighted, in the pavement in the roadway. The car bumped over it. All the passengers were thrown to the top and they got a slight bump. The car was stopped and they got out. This one man said that he had a bad pain in his head, although he had had no injury of any moment. The others were not hurt at all although they seemed to have sustained the same trauma. The man felt badly enough not to continue on his journey but hailed a truck going back to New York and went home.

He did not go to work for a week. He had a pain in his head all that week. On the eighth day he was found unconscious in bed and was taken to hospital by his private doctor who saw him during the next six weeks. During this period his history was that he was running a fever, he was delirious, trying to climb out of bed, he was paralyzed in his right arm and leg, or nearly so, and he was apparently aphasiac and had double choked discs. He recovered from his aphasia, he recovered from his delirium, but he had a very marked hemiplegia. He was totally incompetent afterward.

His case was wrangled through the courts and his attorney got special laws passed in Albany to enable him to try to recover from the State because of this unlighted hole. He was marked down by

everybody as a case of "spate apoplexie." (You know, when you say a diagnosis in German, it sounds more accurate than if you say it in English. He was called a "spate apoplexie," which means delayed apoplexy.)

I agreed to examine the man though I had never seen a case of delayed apoplexy long alive, but such had been the diagnosis for several years. He certainly had hemiplegia very badly but I could not understand how he had apoplexy. The point was that eight days had passed between the very small injury and the apoplexy, and in order to link it up with the injury, they had called it delayed apoplexy.

I said that I did not believe that an apoplexy from injury would occur after a delay of eight days. I could see no pathological foundation for it, but the apoplexy itself was very queer; six weeks of delirium, a high fever, and he had had papilledema. It was a very queer apoplexy.

A search through his hair for a nevus revealed one reason. He had a pink nevus of triangular shape on the left side of his scalp, just over the motor area, which had never been noticed. This made perfectly clear what had happened. Such nevi go right through the layers of the embryo. A very small injury had broken a fragile vessel in the *meningeal* nevus at the time of the bump on the head. It had taken a week of slow leakage, of ingravescent hemorrhage, to produce enough cortical paralysis to give him the appearance of hemiplegia and paralysis of the right arm. He got very large damages, I think, about forty thousand dollars, from the State of New York. The discovery of the nevus linked up the accident to the paralysis eight days later beyond peradventure of a doubt.

I am going now to Bellevue—if I ever get there!—to see a man who was referred, a matter of three weeks ago, with various diagnoses written, after he had a minor head injury, to the effect that he had an organic brain condition because he had a dilated right pupil. His pupil was completely dilated but there was no other neural paralysis symptom whatever and, moreover, it was not just a little dilated but it was wide open. I thought it was a belladonna eye. I could not prove it but the total picture had that lack of

harmony which I spoke of at the beginning of this talk; here was a completely dilated pupil, with no other accident occurring either to the third nerve nucleus or the third nerve. I just could not believe it on technical grounds. I said that he must go into hospital with a night and day nurse, and I looked forward to having a good deal of difficulty in preventing him perhaps from occasionally putting a very small opthalmic disc in his eye over a period of ten days. He refused by advice of his counsel to go to hospital.

But about two weeks ago I realized that there was a God after all! This man was picked up, by an ambulance, with an acute cholecystitis and brought to Bellevue. He had my letter, saying that he was to go into hospital, in his pocket, so he was sent to my ward, with an acute gall bladder. I found him there, by accident, with his dilated pupil and an acute belly. He was turned over to the surgeon for urgent operation and, of course, was necessarily watched. He was not quite himself, under the circumstances, and his pupils today are quite normal. But the fact that he was returned to me, so to speak, on a platter and bound, was very unusual, so I began to believe in the intervention of Divine Providence.

Peter Denker wanted me to speak to you about a case that acquired a certain amount of notoriety here, of a man called Weinstein. I can mention names because it went all through the papers.

This man, who had certificates as to his complete incapacity, for the last ten years had been collecting, I think, something like sixty thousand dollars, on the basis that he had Parkinson's disease. He was only a young man, 43 or 44, and the companies wanted to compound future payments to him. He has been pretty expensive for the last ten years.

He came into my office with colored glasses, walking straight upright, with a big stick. The first thing that struck me was that I had never before seen a case of Parkinson's disease that had to carry a stick. After all, when you begin to think about it, why should a man with paralysis agitans carry a stick? He has lost his automatic associative movements so that that stick does not do him any

good. It would be only a nuisance. That was the first thing that struck me.

I asked him to take off his shoes. His hand was held hard against his ribs, against his side—he was supposed to be a right-sided case—and he was shaking his right hand this way. He bent down and took off his shoes quite quickly with his left hand, and the right hand stayed immobile against his side. There was no slowness of initiating an impulse with his left hand, and the right hand never got going at all.

To cut a long story short, it was perfectly clear that the man was a fake. He used to go out to a camp up in the Catskills where he thought he was pretty safe, and he let loose there and was the life of the party. They sent someone up there to be a guest at the camp, who got very good movies of him and photographs.

I was called down to the Grand Jury to give evidence on medical grounds regarding the difference between a real paralysis agitans and an assumed paralysis agitans. It was very hard to convince a Grand Jury of that, but the upshot of it all was that he was arrested, together with his wife and his daughter, for conspiracy, put in the Tombs and given the evidence that had put him there. He confessed and restored sixty thousand dollars, I understand, of the money. There must have been some sort of a deal made in order to get that money back because I saw in the paper that he had confessed but that he was not being punished because of his physical condition. The report in the District Attorney's files said that his physical condition made it a hazardous thing to bring him to trial.

I felt that I was involved in it a little bit, so I wrote to find out what the physical condition was that jeopardized his life, because I had examined him only a month or so before and I could find nothing at all the matter with him. I got a letter saying that he was a very sick man, that's all. So I think there must have been some "dirty work at the crossroads" to get the sixty thousand dollars back and free him from prison.

A doctor, a man of our own profession, was brought to me last spring who had all the air of idiocy, together with a very

marked tremor. He was supposed to be a paralysis agitans case. He was not. I had him undressed. During the course of the examination, he was just too idiotic for words. He could hardly mumble anything. He kept his mouth open all the time.

He had been going on for about ten years, living down in Florida on one of the Keys. Nobody ever saw him at all. (It seems even our profession is uninteresting to some people. I don't believe I would like to sacrifice my profession to live on a Florida Key.)

He was lying on a couch, undressed, and I noticed a very marked brown stain on his left shin. I said to him, "Doctor, that is a very strange mark. That's a black pigmented mole. Isn't that a malignant thing?" Well, he jerked up so fast to look at it that I knew he wasn't a case of paralysis agitans. You see, he was lying down, and in order to see it, he sat up and he sat up just like a jackknife. I turned in a report and found, on returning from Europe, that I had to certify that he was competent, in order that a guardianship which had been established over him could be removed, and he and his wife agreed to settle on the company's terms. I don't know what the terms were.

A case of a woman, an Italian; another neurologist and I saw her a couple of years ago up at Mount Sinai. She had had a minor injury, had worked and afterwards became psychotic. She was in bed, rolled up like a porcupine, just muttering, and the other neurologist and I could make no diagnosis. She was entirely inaccessible. We said that she was incompetent but I insisted on saying that we did not know the nature of her psychosis.

About a year later, I was surprised to hear that she had been certified as being permanently disabled. She had greatly improved but was still incapacitated, and I was asked to say that she was *permanently* disabled. I said, as I already had said, that I did not know what the matter was with her. Then they told me that the woman was in court and would I see her and examine her. She had recovered sufficiently to be up and she was living over in Jersey with a married sister and her family.

I went to court and found her very neatly dressed, but she could not tell me anything. She could answer questions quite nicely but she could not tell me anything for she could remember nothing. All facts seemed to have gone out of her head. She could not tell me how long she had been in this country nor what age she was. She could not tell me the name of the little town in Jersey where she lived and which she had left two hours before to come to court. She did not know the name of it. She knew that she lived with her sister and her sister's husband. She admitted that.

So I said to her, "You slept with your brother-in-law last night". She said, "I did not"! I said, "You know you did. You shared the same room". She said, "I did not", and went into a furious rage. In order to prove that she did not sleep with her brother-in-law every night, last night or any night, she gave me the plan of the whole flat. She remembered everything. I got her to tell me where the icebox was and all that. I went back to the court room and said that I could not believe that any person who knew the details of her home as accurately as that could forget the name of the place in which she lived, and I thought there was something wrong.

The court ordered the insurance people to have her shadowed. After six months, they were able to show that she was working regularly in another town as a lacemaker. She left her house every morning and got back at a certain hour. She worked under a different name in a different town. She was working as a lacemaker while she was getting, of course, compensation for complete disability. But I doubt if anyone would ever have known about it, until I made her angry by assailing her virtue.

One has to be resourceful, of course, in dealing with these people.

There was a nice case about which some of you may have heard. There was a man sent up to me just after the war, about 1920, from the Marine Hospital. He was sent to the old Neurological Institute on 67th Street. He was supposed to be a Czech, or spoke some language that was so unusual that nobody could communicate with him easily. He alleged that he had lost the use of his

legs in the Navy. He could not stand. The marine officers greatly doubted that but they could not be certain.

I could not be certain either. I knew that he had nothing organically the matter with him. He could move his legs nicely in bed, but every time you put him on his feet he fell down.

I got Dr. Appleby, my house physician at that time, to find the longest stomach tube there was in the hospital. I think this must have been in 1920 or thereabouts because it was during prohibition. It was just after the war but it was during prohibition.

We wrapped a bottle of Scotch in a towel and poured it down the tube. The rubber in the tube prevented him from tasting it, the length of the tube prevented him from smelling it and the towel prevented him from seeing it. Then we removed the tube and left the Scotch. I left the ward and returning in twenty minutes found the place in an absolute bedlam and my friend up out of bed and going from one bed to another saying in a rich Cork brogue, "Sure and this is the foinest hospital Oi've iver been in"!

DR. BROWN—Thank you very much, Dr. Kennedy.

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DR. BROWN—In 1924, Dr. Frank H. Carber, of the Mutual Life of New York, gave us a paper entitled, "The Present Status of Goiter." He has now prepared the paper entitled, "Goiter: Fifteen Years of Underwriting Experience." I know it will be of unusual interest to us to have him tell us of that experience.

GOITER: FIFTEEN YEARS UNDERWRITING EXPERIENCE

FRANK H. CARBER, M. D.

Medical Director, Mutual Life Insurance Company of New York

In a previous communication to this Association (1924) we suggested certain clinical criteria for classification and selection of Goiter cases, and we are now able to submit a report of the results obtained from the practical application of these suggestions to Medical Underwriting.

GENERAL EXPERIENCE

From 1919 to 1934 there were 6,863 Goiter cases submitted for consideration, of which 1,846 cases (26.8%) were declined. The remaining 5,017 cases (73.2%) which were accepted, form the material upon which this study is based.

The average age at issue was 31.7 years. 56.6% were women as compared with 16% in the Company's general experience. There were 28,042 exposures and the mortality experience by lives was 101% of the Company's general experience for the same period. There were 104 Death Claims of which 7 (6.7%) were due to Goiter. (In the general experience of the Company for 1936, .18% of the Death Claims were due to Goiter).

DISABILITY BENEFITS were included in the contracts of 1,858 cases (37%). The average age was 29.8 years. There were 13,365 exposures. The claim experience was 60.7% of the general Disability Claim experience. There were 59 Disability Claims of which 11 (18.6%) were due to Goiter. (In our general claim experience for 1932 to 1934, Goiter was the cause of 0.5% of the total claims).

Of the total 163 claims for both Death and Disability, 18 (11%) were due to Goiter.

CLINICAL SUB-DIVISIONS

Class I—Smooth Symmetrical Non-Toxic Goiters

A—Without Operation

There were 2,135 lives. The average age was 28 years. There were 13,933 exposures and the mortality experience by lives was 90.5%. There were 42 Death Claims of which 2 (4.8%) were due to Goiter.

THE DISABILITY CLAUSE was included in 1,014 of these contracts. There were 7,403 exposures. The claim experience by lives was 63.4%. There were 33 Disability Claims, of which 6 (18.1%) were due to Goiter.

B—With Operation

There were 182 lives. The average age was 32.8 years. There were 1,271 exposures. The mortality experience was 132.7%. There were 8 Death Claims, none of which were due to Goiter.

THE DISABILITY CLAUSE was included in 88 of the above contracts. There were 752 exposures. The claim experience was 64.1%. There were 4 Disability Claims, of which none were due to Goiter.

Class II—Smooth Symmetrical Toxic Goiters

A—Without Operation

There were only 88 lives in this group. There were no deaths.

THE DISABILITY CLAUSE was included in 20 of these contracts. There were no Disability Claims.

B—With Operation

There were 565 lives. The average age was 36.7 years. There were 2,483 exposures. The mortality experience was 119.4%. There were 13 Death Claims, of which 1 (7.7%) was due to Goiter.

THE DISABILITY CLAUSE was included in 130 of these contracts. There were 854 exposures. The claim experience was

74%. There were 5 Disability Claims, of which 2 (40%) were due to Goiter.

Class III—Non-Toxic Nodular or Asymmetrical Goiters

A—Without Operation

There were 261 lives. The average age was 34.3 years. There were 1,221 exposures. The mortality experience was 214%. There were 11 Death Claims of which 2 (18%) were due to Goiter.

THE DISABILITY CLAUSE was included in 56 of these contracts. There were 347 exposures. The claim experience was 81.3%. There were 2 Disability Claims of which 1 (50%) was due to Goiter.

B—With Operation

There were 266 lives. The average age was 34.9 years. There were 1,483 exposures. The mortality experience was 150.6%. There were 10 Death Claims of which none were due to Goiter.

THE DISABILITY CLAUSE was included in 96 of these contracts. There were 719 exposures. The claim experience was 36.6%. There were 2 Disability Claims of which 1 (50%) was due to Goiter.

Class IV—Toxic Nodular or Asymmetrical Goiters

A—Without Operation

There were only 7 lives in this group. There were no Death Claims.

THE DISABILITY CLAUSE was included in 2 of these contracts. There were no Disability Claims.

B—With Operation

There were 143 lives. The average age was 35.7 years. There were 567 exposures. The mortality experience was 93.9%. There were 2 Death Claims of which 1 (50%) was due to Goiter.

THE DISABILITY CLAUSE was included in 19 of these contracts. There were no Disability Claims.

Class V—Goiter, Type Not Clearly Defined

A—Without Operation

There were 912 lives. The average age was 30 years. There were 4,142 exposures. The mortality experience was 60.4%. There were 8 Death Claims, of which none were due to Goiter.

THE DISABILITY CLAUSE was included in 242 of these contracts. There were 1,622 exposures. The claim experience was 34.2%. There were 4 Disability Claims of which 1 (25%) was due to Goiter.

B—With Operation

There were 458 lives. The average age was 35.9 years. There were 2,565 exposures. The mortality experience was 91.4%. There were 10 Death Claims of which 1 (10%) was due to Goiter.

THE DISABILITY CLAUSE was included in 191 of these contracts. There were 1,376 exposures. The claim experience was 86.1%. There were 9 Disability Claims of which 1 (11%) was due to Goiter.

All the above claim ratios for Death or Disability are based upon the Company's general experience for the same period of exposure.

CONCLUSIONS AND COMMENTS

The whole experience given above seems to be large enough to demonstrate that Goiter cases may be selected and insured at standard rates with satisfactory results.

When the experience has been sub-divided into clinical groups, it is found that about half of the exposures appear in *Class I—(Smooth Symmetrical Non-Toxic Goiters, Without Operation).* This experience also seems to be large enough to warrant the conclusion that Simple Goiters (present on examination) may safely be insured at standard rates.

Excess Mortality in unoperated cases was found only in *Class III —(Non-Toxic Nodular or Asymmetrical Goiters)*. The group was not large enough to justify conclusions from the statistical standpoint alone but it is well to remember that the Adenoma is the base from which Thyrotoxicosis, Malignancy or local pressure are particularly likely to develop and great care should be exercised in the selection of this class. Probably these cases are not standard risks.

Among the operated cases, Classes I, II, and III show excess mortality but from a total of 31 deaths, only 1 claim was due to Goiter and it does not seem reasonable to conclude that the impairment should operate against our selection in the cases where the Goiter has been removed.

Below is tabulated in order of frequency the principal causes of death, the percentage of distribution being compared with our General Experience of 1936:

Cases	Cause of Death	Percent of Total	Percent of Total (1936)
16	Cancer (None of Thyroid)	15.4% (Average age 54)	11.3% (Average age 61.5)
15	Organic Heart Disease	14.4% (Average age 52.5)	29.7% (Average age 63.7)
8	Pulmonary Tuberculosis	7.7%	2.7%
8	Iufluenza and Pneumonia	7.7%	6.5%
8	Accidents	7.7%	5.7%
7	Goiter	6.7%	.18%
7	Appendicitis	6.7%	1.5%
5	Cerebral Hemorrhage	4.8%	5.4%
4	Obstetrical	3.8%	2.6%
3	Gastro-Intestinal	2.9%	.9%
2	Insanity	1.9%	1.7%
2	Liver and Gall Bladder	1.7%	1.3%

85 of the total 104 Death Claims are tabulated above. The remaining 19 were all due to different causes, there being only 1 case from each cause.

With the exception of Goiter (which is about 40 times in excess), there seems to be little to indicate that the distribution of causes of death was particularly influenced by the impairment under consideration.

Effect on Mortality Experience From Heart Disease

The condition of "Goiter Heart" is estimated by some Clinicians to constitute 2 to 3% of all cases of heart disease. It is described as a disturbance of cardiac function due to intoxication from excessive thyroid secretion which may terminate in death but leaves no evidence of organic cardiac disease in the event of recovery. Tachycardia may be followed by fibrillation or flutter, acute dilatation or break in compensation but no evidence of permanent cardiac damage can be demonstrated after recovery takes place. This is the effect of acute Thyrotoxicosis and it figures prominently in the proofs of our own Goiter claims for death and disability.

As to *permanent cardiac damage,* it is true that some observers have reported finding slight cardiac enlargement in high percentages of Goiter patients examined in the course of surveys of endemic districts, but this finding is not reflected in the mortality statistics from these regions.

The distribution of chronic heart disease as a cause of death among our Goiter cases was not quite one-half that of our General Experience as tabulated above. There were 15 Death Claims and 1 Disability Claim due to heart disease. Practically all of these cases belong to the coronary, hypertensive or rheumatic groups. None were preceded by evidence of acute "Goiter Heart".

Our own experience does not appear to indicate that Goiter produces permanent cardiac damage.

Dr. Brown—Dr. William O. Pauli, of the Union Central Life Insurance Company, will open the discussion.

Dr. Pauli—I feel that we in the Medical Directors Association are deeply indebted to Dr. Carber for his concise and timely paper on a phase of underwriting which has previously been regarded as a borderline class of risks. It must have entailed an enormous amount of work to classify 5,017 goiter cases, which, no doubt, required careful study of each case with the meager description given by our average medical examiner. Time will not permit repeating the number in each class.

The essayist has given us the meat in a concise report and little can be added to so complete and excellent presentation. I can only comment on his results as compared to other investigations and emphasize certain features.

There were only 104 death claims in 28,042 exposures, and the impairment had no material effect on the cause of death, except the number of deaths due to goiter was increased from .18% (experience of 1936) to 6.7%. There were 7 death claims due to goiter. I would like to ask Dr. Carber whether the number of deaths due to goiter might show a smaller percentage of increase if the essayist had taken the average of a 5-year period for comparison, instead of one year's experience in 1936. Our experience fluctuated considerably. In 1936, we had only 3 death claims due to goiter (.12% of 2,573 claims); in 1935, there were 6 claims due to goiter (.25% of 2,420 deaths); and in 1934, no claims out of 2,486 deaths. Unless the experience of the Mutual Life runs uniform, the percentage of increase of death claims due to goiter would be considerably reduced because of the smaller percentage in 1936. Probably Dr. Carber has found no variation in their mortality for goiter.

I am inclined to reminisce after an experience of 25 years with the Union Central. Looking back to the time previous to 1922, before we issued insurance with a substandard rating, we limited goiter cases to Endowments for not more than 20 years —to mature on or before the age of 60. We required six months to elapse after the removal of a simple goiter. The toxic or exophthalmic goiters were declined. Our reason for limiting this impairment to short Endowments was that the policy would mature before the heart muscle became impaired or any heart complications developed as a result of the toxic effect of the goiter on the heart.

Dr. Carber's investigation shows that the heart is not affected after recovery from the attack. The deaths due to heart disease were not increased during the 15-year period. There were only 15 deaths from heart disease with an average age of 52.5 years, of a total of 101 deaths;— namely, 14.4%. This experience cor-

roborates Dr. Frank Lahey's statement made at the 1932 meeting of this Association: "When a patient is relieved of thyroid toxicity, his heart is just as good as it ever was. There is no evidence to prove that hyperthyroidism produces heart damage." Yet old impressions die hard.

After 1922, we rated non-toxic goiters 125% expected mortality and required 5 years to elapse after operation before we issued standard insurance. All toxic goiters were declined for substandard insurance. In 1929, we began to accept the toxic type of goiter cases without operation after 5 years from recovery of the attack, with a substandard rating of 175%. We also accepted toxic goiters 2 years after operation, with the same rating, and offered standard insurance 5 years after recovery from the operation. The non-toxic adenomatous goiters, or non-toxic hypertrophic, were accepted standard and the toxic adenomas or toxic hyperplastic type were rated the same as toxic goiters. When the M. I. R. was published in 1932, we adopted the ratings of the Joint Committee, except that the adenomas of the thyroid were never rated according to the M. I. R. for adenomas of other glands.

In view of the fact that the Mutual Life does not issue insurance with substandard ratings, it is surprising to note that they accepted 653 toxic type of goiters, 88 with no operation, and 565 with operation. Evidently there was very rigid selection and probably such risks were postponed for a number of years after operation. In view of their favorable experience on the toxic type, I wonder if Dr. Carber will advise us what their rule was—whether they required 5 or even 10 years to elapse after operation or recovery. The mortality of toxic goiters with operation was only 119.4%, even more favorable than the non-toxic group with operation, with a mortality of 132.7%. Both groups were rather small, but their experience is similar to the M. A. M. I. of 1929, which likewise showed a lower mortality for the toxic type of goiter with operation—about one-half of the mortality for simple non-toxic goiters with operation.

Dr. Jimenis, of the Metropolitan, made a report at the 1932 meeting, showing their experience on goiter, including the issues of 1912 to 1928. There were 12 deaths due to goiter among females, with non-toxic goiter accounting for over 11 times the normal number; and the deaths due to organic diseases of the heart were less than the normal number of expected deaths from heart disease.

In the 1936 investigation of the Joint Committee of issues from 1920 to 1934 based on policies, there were 20,000 exposures due to toxic goiters with operation, and the mortality was only 89% with 76 deaths. This includes both the standard and the sub-standard, and in only 20 of these deaths (26%) was it indicated whether the goiter was nodular or diffuse.

The unfavorable group in the Mutual Life series was the non-toxic nodular, or asymmetrical, goiters. There were 21 claims out of 427 lives, 214% mortality on the cases without operation, and 150.6% mortality with operation. This is contrary to expectations, especially the group with operations, as non-toxic adenomas after removal are given favorable prognosis by most surgeons. The group was too small to warrant more than a caution in the selection of this group, as only 2 of the 21 deaths were due to goiter, and no deaths were due to goiter in the group with operation.

From an insurance viewpoint, we are not concerned with the adolescent type of goiter. Over 90% of the adolescent goiters disappear spontaneously with maturity. Those that are nodular are more liable to persist at middle age.

The improvement in the mortailty of toxic goiters, I believe, is due to the lowered operative mortality resulting from operations for thyroidectomy. It is now 15 years since surgeons began to use iodine in the preparation of patients for operation on the thyroid. The Lahey Clinic, of Boston, shows an operative mortality of 3.5% for toxic cases with thyroid crisis, and .85% for all types of goiter. The mortality rate is almost the same in all first-grade thyroid clinics.

We are all very much interested in the disability claim experience on goiter cases. The Mutual Life is to be congratulated upon their favorable disability experience with 1,858 cases, as only 12 were disabled due to goiter. They had only 60.7% of the normal disability claim experience on these goiter cases. We, like most companies, were too liberal in granting disability benefits to all cases approved at standard rates. We had 25 claims for disability due to goiter, of a total of 2,528 approved claims. I reviewed the disability claims on account of goiter and found that none of them had a history or a record of goiter at the time we approved the risk. Our present practice is to issue Waiver of Premium Disability Clause only after 5 years had elapsed from the date of operation or recovery from toxic goiters. The average duration of total disability on account of goiter in our experience was 3-¾ years, and 5 of the 25 claims were receiving payments from 8 to 10 years and are still collecting monthly payments. All of our claims were due to toxic goiters, or exophthalmic goiters, except one case of thyroiditis, which was complicated by arthritis. The average age of our disability cases was 29.7 years, and 10 of the 25 claims were on female lives. We place on the books about 70 policies a year, where the medical examination reveals either a history of goiter or a goiter on examination.

In closing my discussion, I believe we can safely disregard any danger of heart damage from goiter and that we can be more liberal with our selection of toxic goiters than we have been in the past.

DR. BROWN—Dr. George McCreight, of the Bankers Life Company, will continue the discussion.

DR. MCCREIGHT—Dr. Carber's report on their experience in selection of thyroids is most significant. It indicates that, with proper classification, goiters are safely insurable at standard rates or on some substandard basis. The very recent recommendation of the Committee on Medical Impairments proves this point. A serviceable classification was outlined by Carber in a paper presented before this Association in 1924. An efficient questionnaire helpful in classifying goiters was introduced.

Clinical factors must always be kept in mind if we are to successfully classify goiters. Dr. Carber's figures speak for themselves and, therefore, in the following discussion, clinical ideas are presented which I hope will be helpful.

It is of additional interest that the years covered by his experience, that is, from 1919 to 1934, include the period in which the greatest advance in thyroid work has been done.

Operative mortality alone has dropped from around 4% to less than eight-tenths per cent. during this time.

The prophylactic uses of iodine in goiter districts have shown its good effect by materially reducing goiters in these endemic regions. Although the indiscriminate use of iodine is controversial, nevertheless, statistics have proven that injurious effects are minimum and that without question it has been a most forward step in the prevention of the simple type of goiter in the areas where it is used.

It is an accepted fact that the public is becoming more goiter-conscious. There is not the former fear connected with goiter surgery and the patient is presenting himself to the doctor with less hesitation. With our modern conception of thyroid, the cases are worked out scientifically. Proper diagnoses are being made and if surgery is indicated, the patient comes to the operating table before any definite damage from over-thyroid activity has been done.

Probably the greatest advances in thyroid work have been accomplished through the use of the basal metabolism test, the knowledge gained from Plummer's classification, and his most excellent work in determining the effect of iodine in hyperplastic thyroids. The year 1922 marks the introduction of the use of iodine in preparing for operation patients with exophthalmic goiters. At the same time it marks the beginning of a further advance in the diagnosis of goiters and more general recognition of the importance of correct differential diagnosis.

Plummer, by his clinical observations which resulted in his separating exophthalmic goiter from hyperfunctioning adenomas, established fundamental characteristics of the former condition.

Evidences of hyperthyroidism were present in both conditions but in the exophthalmic type there were also present certain phenomena that could not be produced by the feeding of thyroid substances; namely, exophthalmos and other ocular changes, the characteristically useless but purposeful movements, a psychic state indicative of cerebral intoxication and the gastro-intestinal crises. The characteristic effect of iodine is to abolish or stop the progress of these specific phenomena.

Thus the use of iodine as a diagnostic agent is essential in cases where these characteristic symptoms are not definite. A drop in basal metabolic rate, a lowering of a fast pulse, a reduction of nervous symptoms and an improvement of other symptoms would indicate a true exophthalmic goiter.

As a diagnostic method, iodine has been used with excellent success and is of aid in ruling out adenomas, neurocircular asthenias, hypertensive diseases and heart conditions such as auricular fibrillation, irregularities in pulse, tachycardia and other organic heart diseases. Iodine will lower the basal metabolic rate in hyperthyroidism, slows the pulse and increases the sense of well-being and causes gain in weight. This is not usually true in the neurocirculatory or other allied diseases which might be confused with the thyroid.

Prior to the pre-operative use of iodine, the Mayo Clinic experienced a death rate of 1 to 2% before the patients could be prepared for operation. Before Plummer proved the effectiveness of iodine treatment in this type of goiter, the impression prevailed among the members of the medical profession that iodine was definitely contraindicated in hyperthyroid cases.

The typical case of exophthalmic goiter presents such striking signs that it is not difficult to make a diagnosis. The common picture of extreme nervousness, exophthalmos, tremor, excessive sweating and tachycardia are so familiar that they do not need emphasis. It is in the earlier stages where these phenomena are not so marked that a true hyperthyroidism may be easily overlooked. There are certain signs and symptoms which always call for a thorough investigation of the functions of the thyroid gland.

Any unexplained loss of weight, especially simultaneously with a normally increased appetite, should suggest hyperthyroidism. Increased pulse pressure in the absence of aortic insufficiency is a significant sign. Demonstrable loss of strength is a symptom. Tachycardia, of course, so frequently will suggest such a variety of conditions that it is of less significance than other signs. However, if it is constant it should be considered in the diagnosis of hyperthyroidism.

Many patients with cardiac decompensation are suffering from hyperthyroidism and in fact it is the latter condition which overloads the heart to the point of decompensation. Nervous exhaustion may be a difficult factor in differential diagnosis since the nervous manifestations of fatigue may simulate a mild hyperthyroidism. These cases not uncommonly have an elevated metabolic rate. This is caused by their inability to relax sufficiently at the time the test is made. Frequent repetition of the test will usually rule out this condition.

If diagnosis of thyroid disease is so obscured as to preclude a positive opinion, careful observation during the period in which iodine is administered often will clear up the diagnosis. It is usually necessary to give iodine for 7 or 8 days to establish a diagnosis.

Patients with essential hypertension frequently present many manifestations of hyperthyroidism, including an increased basal metabolic rate. Rest in bed will occasionally result in a normal rate and the effect of iodine is also useful in differentiating these cases but is of less value than in cases of nervous exhaustion.

Because of the significance of the basal metabolism test, its use has come into prominence during the period in which Dr. Carber has made his survey. It has become a reliable means of diagnosing hyperthyroidism if the test has been accurately made and on a reliable machine. The results of any basal metabolism test should always be adjudged in connection with a well-worked out clinical case. In questionable cases, a series of basal metabolic rates should be taken in order to arrive at a definite conclusion.

Patients with cardiac decompensation sufficient to produce dyspnea will have increased basal metabolic rates. It has been demonstrated that the increased rate is due solely to the dyspnea and as soon as dyspnea disappears under appropriate treatment the rate falls to normal, except in cases where there is hyperthyroidism.

It is, of course, important to know in all cases whether hyperthyroidism is associated with cardiac decompensation. Often the patient does not show evidence of enough cardiac injury to explain the decompensation. Increased pulse pressure may be the most suggestive sign. Unexpected loss of weight before the development of decompensation should make one suspect the thyroid. Auricular fibrillation always suggests presence of an overactive thyroid.

There are certain characteristics evidenced in the exophthalmic goiter that are not seen in the cases of hyperfunctioning adenomatous goiters. Any effort to diagnose exophthalmic goiters more accurately must come from a study of these characteristics and repeated observation of patients with these characteristics in mind.

It seems to me as underwriters that we should place goiters in the classification originally outlined by Dr. Carber. We can do this only by the complete co-operation of our examiners in the field in obtaining full and detailed clinical history, including the special laboratory reports supplemented by a summary of the attending physician's records.

I think you will agree that we get excellent information from our larger clinics and from men who are specializing in goiter work. Our big difficulty comes from the average practitioner, who is not equipped or is negligent in working out his goiter cases according to our modern understanding of goiters.

It is our problem to obtain all available information regarding these cases. Such information should include a diagnosis as to the type of goiter. This also should include a complete medical history with particular attention given to weight loss, nervousness, fast pulse, exophthalmos, basal metabolic rates, therapeutic use of iodine and possible iodine examination of the blood. And, in addition, the information regarding surgery, the length of time symp-

toms had been experienced prior to operation, the report of a competent pathologist as to the type of goiter removed, the length of time following the operation, whether it has been necessary to operate by the one stage or multiple stage plan and the result of the basal metabolism test, preceding and during a period of from one to two years following thyroidectomy.

After obtaining the necessary information, the problem is ours to solve in accordance with our knowledge, judgment and experience.

DR. BROWN—Dr. Carber, will you close the discussion?

DR. CARBER—In his discussion, Dr. Pauli referred to the average distribution of goiter deaths in the experience of the company, that is, the general experience, asking if it was uniform. By asking this question, he brought out an error in the original paper. The figure was .18 average for 1936. Instead of that, I find that the average distribution of goiter deaths in 1936 was .11. The figure which was given, .18, is the average of the years 1931, 1932, 1933, 1934 and 1935. The variation in 1931 was .33; in 1932, it was .19; in 1933, it was .09; in 1934, it was .17; and in 1935, it was .13.

Dr. Pauli also asked the question as to criteria of selection of toxic goiters which have been operated. We take them three years after operation, if there is no evidence of any of the remaining effects of thyroid intoxication, with the exception of exophthalmos. Exophthalmos usually continues in a high percentage of these cases.

He also spoke of the mortality of operated cases. There were 1,600 operated cases and the mortality of these 1,600 was 116-plus per cent. Why the mortality in operated cases is higher than the mortality in unoperated cases I cannot answer. That same fact is true in other impairments, notably duodenal ulcer. The mortality in the unoperated cases is usually better than in the operated cases. It is also true in renal colic as compared to the cases where a stone was definitely found and operated.

In conclusion, then, I want to thank Dr. Pauli and Dr. McCreight for their discussion, which showed that they had given considerable

time and consideration to the paper which was submitted to them.

* * * * * *

DR. BROWN—We have now come to the end of our program of scientific papers. Lunch will be served shortly under the same circumstances as yesterday. I thank you for the honor of having been your President. You have been kind, patient and sympathetic. To all those who have assisted me during the past year and during this meeting, I extend my sincere thanks, and state that without them and without the help which they have unstintingly given, I could have done very little.

You have elected a new President; the Association is to be congratulated upon the choice which it has made. Will Dr. Samuel B. Scholz, who is now President, take the chair.

DR. SCHOLZ—This is a momentous occasion for me, and it is with great pleasure that I have the privilege of addressing you, and expressing to you individually and collectively my appreciation of the confidence you have placed in me, and the honor conferred, in your election of me as President of this scientific organization which has accomplished so much in life insurance medicine.

Dr. Keating of the Penn Mutual Life Insurance Company, in May, 1889, invited a group of Medical Directors to join him in a conference to discuss procedures for a fuller co-operation between Medical Directors, and to form a medical group for purposes of a more intimate acquaintanceship between Medical Directors and for an exchange of thought concerning life insurance medicine. From this committee developed the Association of Life Insurance Medical Directors of America.

The responsibility for the success of our Association has rested not so much upon the elective officers as upon the delightful, co-operative spirit of the entire membership.

This meeting has been an intensely interesting and a very profitable one. The retiring President has my felicitations; and

with your help, Gentlemen, we shall hope for our next session a number of equally instructive, scientific theses.

During my tenure of office I shall feel deeply obligated to you who make suggestions for the furtherance of the objectives of our Association.

Dr. Rowley, I believe you have something to present to the Association?

DR. ROBERT L. ROWLEY (Phoenix Mutual Life Insurance Company)—Before this splendid meeting comes to an official close, I should like to say just a word attesting to the high quality of this meeting, from start to finish, in every detail. Our program has been excellently prepared; the papers have been very instructive and valuable to us; the prepared discussions have been of the highest order, and we have all enjoyed and profited very much from the addresses given to us by our invited guests.

The office of President has been filled in the past year in a most notable manner by our retiring President and I know the feeling in the minds of each one of you and in the minds of those who may not now be in the audience but who have had the good fortune to attend some of the meetings, and in expressing the feeling which I know you all would like to express, I offer this resolution:

"RESOLVED: That this Association extend its thanks and appreciation for the admirable manner in which Dr. Brown has administered his office as President, as well as for the outstanding quality of his program."

* * * * * *

This motion was carried unanimously. Whereupon, as there was no other business before the Association, the meeting was adjourned.

During the past year, five members of our Association have died. The following testimonials to their memory have been prepared by representatives of the Association.

DR. CHARLES D. ALTON

1845–1937

It is with the deepest regret that I bring to your attention the death on January 8 of Dr. Charles D. Alton, Medical Referee of the Connecticut Mutual Life Insurance Company of Hartford, and a member of this Association.

Dr. Alton was born in Kenosha, Wisconsin, May 9, 1845, a son of Condé Raguet Alton and Carolan Esther (Turner) Alton. He secured his preliminary education at Phillips Exeter Academy, graduating in 1866. His medical training was attained at Bellevue Hospital Medical College, from which institution he graduated in 1875.

During his long residence in Hartford, he was not only active professionally, but also had a lively interest in civic affairs. He was instrumental in the formation of the Open Hearth Mission, and served as its President for several years. Dr. Alton was also President of the Hartford County Medical Association in 1912, and served as President of the Hartford Medical Society in 1913. For ten years he served that body as Treasurer. He was also Vice President of the American Climatological Association. Dr. Alton was a member of the Twentieth Century Club, and a former President; a member of Trinity Church, the Connecticut Historical Society and the Drama Society of Hartford.

He leaves two children, Mary Brace Horne and Carolan Hayward.

All of us who have had the privilege of knowing Dr. Alton feel a deep and personal loss in his passing from our midst.

WILLIAM DENISON MORGAN, M. D.

1850–1936

Dr. Morgan was a member of this association for more than 42 years—from June 13, 1894, until his death on December 17, 1936.

He was born in Hartford on November 20, 1850; was a graduate of Trinity College in 1872, and of the College of Physicians and Surgeons, Columbia University in 1876. Following his internship at the New Haven Hospital he studied for a year in Germany.

Returning to Hartford in 1878 he engaged in the practice of his profession and soon established himself among the leaders in medical circles in the city and in the state.

In 1894 he became a member of the medical department of the Phoenix Mutual Life Insurance Company; shortly afterwards was appointed Assistant Medical Director, and in 1906 he became Medical Director and held the title until his death.

For many years he was a regular attendant at the meetings of this association and his keenness of mind, his forceful personality and great sense of humor will continue to abide in the memories of the older members.

The following tribute is reprinted from an editorial in the Hartford Courant:

> "Dr. William Denison Morgan, 'Uncle Billy', to his many friends, lived most of his eighty-six years in Hartford. He could trace his ancestry to the early beginnings of the Connecticut colony, but pride of descent was no part of his cosmopolitan makeup, however much he may have valued it. During the years of his active practice he contributed liberally of his time and abounding energy to various institutions within the sphere of his profession. To him the Hartford Hospital, in particular, owes a deep debt of gratitude. Members of its board of trustees could come and go, but he remained on; for nearly thirty years he was chairman of the executive committee, and on his retirement he carried the title of honorary chairman.

"His interest in the orphan asylum and in the State Hospital for the Insane at Middletown far surpassed his professional obligations to them. Kind and charitable, he fitted well Voltaire's definition of a physician as an estimable man, 'who, having studied nature from his youth, knows the properties of the human body, the diseases which assail it, the remedies which will benefit it, exercises his art with caution, and pays equal attention to the rich and the poor'.

"A familiar figure at the Hartford Club, where in his later years he lived, his jovial nature found abundant expression. No matter what turn the conversation might take, the topic seemed always familiar to him. His love of the classics had given him a rich background. He had read about everything worth reading, and he followed current events, especially in the field of politics and economics, with a full understanding of their significance. His was a keen and penetrating mind, tempered with an appreciation of humor that made his company eagerly sought. Age did not dim his eye or diminish his enjoyment of life. If ever a man possessed the art of growing old gracefully and belying his years, it was Dr. Morgan, and many there will be to mourn him."

DR. THOMAS HAWLEY ROCKWELL

1865–1936

Dr. Thomas Hawley Rockwell, for twenty years Medical Director of the Equitable Life Assurance Society of the United States, died November 2, 1936, at Summit, New Jersey, at the age of seventy-one. His health had been failing for some months.

Dr. Rockwell was born at Warsaw, Illinois, September 18, 1865, of pioneer American stock. He spent his early boyhood at Junction City, Kansas, and graduated from the University of Kansas where he was a Phi Gamma Delta. He obtained his medical training at the old College of Physicians and Surgeons at New York before it became affiliated with Columbia University. His

first medical work, after his interneship at a charity hospital, was as a surgeon on the Costa Rica Railroad, which was then building from Port Limon to San Jose. He became an examining physician with the Equitable Life Assurance Society at Chicago in 1890, and in 1900 was appointed the Society's representative in London, England, for its European affairs. In 1916 he was made the Chief Medical Director of the Equitable, which post he held until June 1936, when his failing health forced him to retire.

When he was President of the Association of Life Insurance Medical Directors, 1918-1919, in addition to the annual meeting, he arranged a joint meeting of the Association and the Actuarial Society, which was quite a success. He took an active part in the Association not only in presenting papers but in discussions, and served on many of its important committees.

A man of sterling character and attractive personality, he was much beloved by his associates, as well as by all those who had the privilege of serving him as Medical Director of the Equitable Life. To them all his death came as a great personal loss.

DR. EDWARD KING ROOT

1857-1937

Dr. Edward K. Root, Honorary Medical Director of the Aetna Life Insurance Company, died at his summer home in Fenwick on August 12, 1937.

Dr. Root had been associated with the Aetna Life Insurance Company since 1899 as Medical Director.

Always civic-minded, he held a number of important positions in the state and city in which he lived, being a member of the State Board of Health from 1899 to 1917 and serving as its President from 1909 to 1917, at which time the State Board of Health became the Public Health Council and he served as its Secretary until 1929 when he resigned. He was Chairman of the Hartford Board of Health from 1899 to 1902 and was associated with that Board until 1913. He was a visiting physician to the Hartford Hospital until 1923 when he retired to give his full time

to the Aetna Life. While filling these various positions he was responsible for the building of the Isolation Hospital, now a part of the Municipal Hospital, and also Wildwood Sanitarium, the tubercular branch of the Hartford Hospital.

He also found time to act as a Director of the Hartford Electric Light Company, the Connecticut Power and Light Company, and the Collins Company, all of Hartford.

His medical training was an M.D. from the University of the City of New York in 1879, supplemented by work in the Rotunda Hospital in Dublin and study on the Continent. He was a member of the City, County, State, and American Medical Associations; also a non-resident member of the New York Academy of Medicine.

He was President of the Medical Directors' Association in 1913 and was always interested in the organization and its aims.

His personality was delightful. Always of a pioneering spirit. Always ready and interested in any new developments. At the same time his judgment and experience were always in the background to aid him in forming an opinion concerning the use of any new apparatus or formula.

His paper on "Family History in Tuberculosis" which was given as his presidential offering is worth referring to.

He was a delightful conversationalist and enjoyed nothing better than an evening with his friends, discussing and reminiscing.

To his assistants in the Company he was always a source of inspiration and a tremendous aid in their education and development, smoothing out difficulties with a rare, whimsical touch which endeared him to his associates and friends. His influence in the organization will always be felt through the passing years.

He is survived by his wife, Era Steele Root, and a step-daughter, Mrs. Morgan G. Bulkeley, Jr.

DR. DeWITT SMITH

1888–1937

Dr. DeWitt Smith died suddenly, following a heart attack, October 2, 1937.

Dr. Smith was born July 2, 1888, at Eaton, Tennessee. He attended Vanderbilt University, where he received his M. D. degree in 1913. After practicing for a short while in Trenton, Tennessee, he moved to Dallas in 1917. During the World War he served overseas as a captain in the medical corps attached to the Mayo Clinic group. Returning from the war, he resumed his practice in Dallas and soon became chief medical examiner for Southwestern Life Insurance Company. In January, 1926, he was made Medical Director of that Company, which position he held at the time of his death.

Dr. Smith was president of the Vanderbilt University Alumni Association of Dallas, a member of the American Medical Association, the Dallas County Medical Society, Southern Clinical Society, Delta Kappa Epsilon fraternity, and other prominent medical, business, and social organizations.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
A. W. Barrows, M. D.	Hartford, Conn.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Robert J. Blanchard, M. D.	Winnipeg, Man.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York City
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Henry Colt, M. D.	Pittsfield, Mass.
Thomas C. Craig, M. D.	New York, N. Y.
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Landon Carter Gray, M. D.	New York, N. Y.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Joseph B. Hall, M. D.	Hartford, Conn.
Edward H. Hamill, M. D.	Newark, N. J.

DECEASED MEMBERS

William W. Hitchcock, M. D.	Los Angeles, Calif.
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Henry H. Hutchison, M. D.	Toronto, Canada
Lefferts Hutton, M. D.	New York City
Phineas H. Ingalls, M. D.	Hartford, Conn.
Arthur Jukes Johnson, M. D.	Toronto, Ont.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.
Henry P. Lyster, M. D.	Detroit, Mich.
Charles N. McCloud, M. D.	St. Paul, Minn.
Francis A. McGreen, M. D.	New York, N. Y.
Lewis McKnight, M. D.	Milwaukee, Wis.
Thomas H. McMahon, M. D.	Toronto, Ont.
Elias J. Marsh, M. D.	Paterson, N. J.
Henry A. Martelle, M. D.	Hartford, Conn.
Allison Maxwell, M. D.	Indianapolis, Ind.
Archibald Mercer, M. D.	Newark, N. J.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William D. Morgan, M. D.	Hartford, Conn.
John P. Munn, M. D.	New York, N. Y.
William Natress, M. D.	Toronto, Ont.
Edwin M. Northcott, M. D.	Portland, Me.
Brace W. Paddock, M. D.	Pittsfield, Mass.
Frank K. Paddock, M. D.	Pittsfield, Mass.
William A. Peterson, M. D.	Chicago, Ill.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James T. Priestly, M. D.	Des Moines, Iowa
Oliver P. Rex, M. D.	Philadelphia, Pa.

DECEASED MEMBERS

Thomas H. Rockwell, M. D.	New York, N. Y.
Edward K. Root, M. D.	Hartford, Conn.
James F. W. Ross, M. D.	Toronto, Ont.
Gurdon W. Russell, M. D.	Hartford, Conn.
George R. Shepherd, M. D.	Hartford, Conn.
De Witt Smith, M. D.	Dallas, Texas
George S. Stebbins, M. D.	Springfield, Mass.
George S. Strathy, M. D.	Toronto, Ont.
Melancthon Storrs, M. D.	Hartford, Conn.
Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
James Thorburn, M. D.	Toronto, Ont.
James D. Thorburn, M. D.	Toronto, Ont.
William Thorndike, M. D.	Milwaukee, Wis.
Paul E. Tiemann, M. D.	New York, N. Y.
Harry Toulmin, M. D.	Philadelphia, Pa.
Frank L. Truitt, M. D.	Indianapolis, Ind.
Henry Tuck, M. D.	New York, N. Y.
John S. Turner, M. D.	Dallas, Texas
Joseph P. Turner, M. D.	Greensboro, N. C.
S. Oakley Van der Poel, M. D.	New York, N. Y.
Charles A. VanDervoort, M. D.	Philadelphia, Pa.
A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. VanDyck, M. D.	New York, N. Y.
Aaron C. Ward, M. D.	Newark, N. J.
Wm. Perry Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Ont.
William E. H. Wehner, M. D.	Philadelphia, Pa.
Ernest A. Wells, M. D.	Hartford, Conn.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
A. H. Whitridge, M. D.	Baltimore, Md.
George Wilkins, M. D.	Montreal, Que.
Thomas H. Willard, M. D.	New York, N. Y.
Gordon Wilson, M. D.	Baltimore, Md.
G. S. Winston, M. D.	New York, N. Y.
Harry P. Woley, M. D.	New York, N. Y.
Albert Wood, M. D.	Worcester, Mass.
Green V. Woolen, M. D.	Indianapolis, Ind.
John C. Young, M. D.	Detroit, Mich.
Joseph C. Young, M. D.	Newark, N. J.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

Edwin H. Allen, M. D.	John Hancock, Boston, Mass.
George E. Allen, M. D.	National Life, Montpelier, Vt.
Hiram H. Amiral, M. D.	State Mutual Life, Worcester, Mass.
Harry B. Anderson, M. D.	Imperial Life, Toronto, Canada.
Robert B. Andrews, M. D.	Massachusetts Mutual, Springfield, Mass.
Thomas D. Archibald, M. D.	North American, Toronto, Canada.
Edward M. Armstrong, M. D.	Mutual Life, New York City.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
Malcolm O. Austin, M. D.	West Coast Life, San Francisco, Calif.
J. Albert Avrack, M. D.	United States Life, New York City.
Henry A. Baker, M. D.	Kansas City Life, Kansas City, Mo.
Henry A. Bancel, M. D.	Mutual Life, New York City.
G. Holbrook Barber, M. D.	Manhattan Life, New York City.
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
William B. Bartlett, M. D.	John Hancock, Boston, Mass.
John T. J. Battle, M. D.	Jefferson Standard, Greensboro, N. C.
Edgar W. Beckwith, M. D.	Equitable Life Assurance, New York City.
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.

Roland A. Behrman, M. D.	John Hancock, Boston, Mass.
Maurice B. Bender, M. D.	Guardian Life, New York City.
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
Frank M. Beresford, M. D.	Provident Mutual, Philadelphia, Pa.
Charles C. Berwick, M. D.	Metropolitan Life, New York City.
Edward B. Bigelow, M. D.	State Mutual Life, Worcester, Mass.
Cecil C. Birchard, M. D.	Sun Life, Montreal, Canada.
Wilton F. Blackford, M. D.	Commonwealth Life, Louisville, Ky.
David N. Blakely, M. D.	New England Mutual, Boston, Mass.
Harold E. Bogart, M. D.	New York Life, New York City.
William Bolt, M. D.	New York Life, New York City.
Earl C. Bonnett, M. D.	Metropolitan Life, New York City.
J. Thornley Bowman, M. D.	London Life, London, Canada.
William M. Bradshaw, M. D.	Mutual Life, New York City.
Frederick G. Brathwaite, M. D.	Equitable Life Assurance, New York City.
Albert W. Bromer, M. D.	Metropolitan Life, New York City.
Chester T. Brown, M. D.	Prudential, Newark, N. J.
Frederick R. Brown, M. D.	New England Mutual, Boston, Mass.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Benjamin F. Byrd, M. D.	National Life & Accident, Nashville, Tenn.
Joseph T. Cabaniss, M. D.	Travelers, Hartford, Conn.
Edward J. Campbell, M. D.	New York Life, New York City.
Frank H. Carber, M. D.	Mutual Life, New York City.

Laurence D. Chapin, M. D.	Massachusetts Mutual, Springfield, Mass.
Charles L. Christiernin, M. D.	Metropolitan Life, New York City.
Charles P. Clark, M. D.	Mutual Benefit, Newark, N. J.
Augustus D. Cloyd, M. D.	Woodmen of the World, Omaha, Neb.
Edwin A. Colton, M. D.	National Life, Montpelier, Vt.
Henry W. Cook, M. D.	Northwestern National, Minneapolis, Minn.
Parker M. Cort, M. D.	Aetna Life, Hartford, Conn.
Donald B. Cragin, M. D.	Aetna Life, Hartford, Conn.
Hugh W. Crawford, M. D.	Columbian National, Boston, Mass.
Hamilton C. Cruikshank, M. D.	Manufacturers Life, Toronto, Canada.
Robert M. Daley, M. D.	Equitable Life Assurance, New York City.
Joe E. Daniel, M. D.	Great Southern Life, Houston, Texas.
William L. Davis, M. D.	John Hancock, Boston, Mass.
Harold D. Delamere, M. D.	Crown Life, Toronto, Canada.
Peter G. Denker, M. D.	Equitable Life Assurance, New York City.
Ernest J. Dewees, M. D.	Provident Mutual, Philadelphia, Pa.
Edwin G. Dewis, M. D.	Prudential, Newark, N. J.
Thomas H. Dickson, M. D.	Minnesota Mutual, St. Paul, Minn.
Edward S. Dillon, M. D.	Penn Mutual, Philadelphia, Pa.
Harold W. Dingman, M. D.	Continental Assurance, Chicago, Ill.

William W. Dinsmore, M. D.	Travelers, Hartford, Conn.
Percy G. Drake, M. D.	Travelers, Hartford, Conn.
Joseph B. Drummond, M. D.	Union Mutual, Portland, Me.
James H. Dunkley, M. D.	Shenandoah Life, Roanoke, Va.
John T. Eads, M. D.	Penn Mutual, Philadelphia, Pa.
O. M. Eakins, M. D.	Reliance Life, Pittsburgh, Pa.
Joseph E. Engelson, M. D.	Mutual Life, New York City.
Francis C. Evers, M. D.	New York Life, New York City.
William G. Exton, M. D.	Prudential, Newark, N. J.
James G. Falconer, M. D.	North American Life, Toronto, Canada.
Raymond K. Farnham, M. D.	Metropolitan Life, New York City.
Robert H. Feldt, M. D.	Northwestern Mutual, Milwaukee, Wis.
Haynes H. Fellows, M. D.	Metropolitan Life, New York City.
John Ferguson, M. D.	Excelsior Life, Toronto, Canada.
William E. Ferguson, M. D.	Excelsior Life, Toronto, Canada.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.
Robert A. Fraser, M. D.	New York Life, New York City.
Edward M. Freeland, M. D.	New York Life, New York City.
Harold M. Frost, M. D.	New England Mutual, Boston, Mass.
Homer Gage, M. D.	State Mutual, Worcester, Mass.

F. Irving Ganot, M. D.	Prudential, Newark, N. J.
Kenneth Gardner, M. D.	Reliance Life, Pittsburgh, Pa.
Edson E. Getman, M. D.	New York Life, New York City.
Alexander D. Ghiselin, M. D.	Equitable Life Assurance, New York City.
Henry W. Gibbons, M. D.	California Western States, Sacramento, Calif.
Ralph T. Gilchrist, M. D.	Northwestern Mutual, Milwaukee, Wis.
Robert A. Goodell, M. D.	Phœnix Mutual, Hartford, Conn.
Harry W. Goos, M. D.	Home Life, Philadelphia, Pa.
John K. Gordon, M. D.	Sun Life, Montreal, Canada.
Angus Graham, M. D.	London Life, London, Canada.
LeRoy C. Grau, M. D.	Travelers, Hartford, Conn.
Robert J. Graves, M. D.	United Life & Accident, Concord, N. H.
Clarence M. Grigsby	The Praetorians, Dallas, Texas.
Frank L. Grosvenor, M. D.	Travelers, Hartford, Conn.
Frederick W. Hagney, M. D.	Mutual Benefit, Newark, N. J.
Llewellyn Hall, M. D.	Phœnix Mutual, Hartford, Conn.
F. Tulley Hallam, M. D.	State Life, Indianapolis, Ind.
George A. Harlow, M. D.	Northwestern Mutual, Milwaukee, Wis.
Frank Harnden, M. D.	Berkshire Life, Pittsfield, Mass.
Walter C. Hausheer, M. D.	Prudential, Newark, N. J.
William D. Heaton, M. D.	New York Life, New York City.

Ernest M. Henderson, M. D.	Confederation Life, Toronto, Canada.
Carroll E. Herron, M. D.	Ohio State, Columbus, Ohio.
William L. Hilliard, M. D.	Equitable Life, Waterloo, Canada.
Daniel W. Hoare, M. D.	Penn Mutual, Philadelphia, Pa.
William W. Hobson, M. D.	Reliance Life, Pittsburgh, Pa.
Donald C. Hoffman, M. D.	Metropolitan Life, New York City.
Chauncey O. Hollinger, M. D.	Prudential, Newark, N. J.
Byam Hollings, M. D.	John Hancock, Boston, Mass.
Charles E. Homan, M. D.	Connecticut Mutual, Hartford, Conn.
Joseph C. Horan, M. D.	Metropolitan Life, New York City.
John L. Humphreys, M. D.	Reliance Life, Pittsburgh, Pa.
James H. Humphries, M. D.	Home Life, New York City.
Ross Huston, M. D.	Bankers Life, Des Moines, Iowa.
William G. Hutchinson, M. D.	American Life, Detroit, Mich.
Washington C. Huyler, M. D.	Mutual Life, New York City.
William G. Hyde, M. D.	Northwestern Mutual, Milwaukee, Wis.
Charles E. Iliff, M. D.	Western and Southern, Cincinnati, Ohio.
J. Grant Irving, M. D.	Aetna Life, Hartford, Conn.
Charles B. Irwin, M. D.	North American, Chicago, Ill.
Walter A. Jaquith, M. D.	Columbus Mutual, Columbus, Ohio.
Frank L. B. Jenny, M. D.	Federal Life, Chicago, Ill.
Albert O. Jimenis, M. D.	Metropolitan Life, New York City.
Albert E. Johann, M. D.	Bankers, Des Moines, Iowa.

George E. Kanouse, M. D.	Prudential, Newark, N. J.
Edward A. Keenleyside, M. D.	Connecticut General, Hartford, Conn.
Herbert B. Kennedy, M. D.	Woodmen of the World, Omaha, Neb.
Frank W. Kenney, M. D.	Capitol Life, Denver, Colo.
Harry B. Kidd, M. D.	Metropolitan Life, New York City.
Oscar W. King, M. D.	Equitable Life Assurance, New York City.
Henry B. Kirkland, M. D.	Prudential, Newark, N. J.
Robert J. Kissock, M. D.	Metropolitan Life, New York City.
Edward R. H. Kurz, M. D.	Prudential, Newark, N. J.
William P. Lamb, M. D.	Prudential, Newark, N. J.
Anthony J. Lanza, M. D.	Metropolitan Life, New York City.
Ivan C. Lawler, M. D.	New York Life, New York City.
Linford H. Lee, M. D.	Pacific Mutual, Los Angeles, Calif.
John M. Livingston, M. D.	Mutual Life, Waterloo, Canada.
G. Carroll Lockard, M. D.	Maryland Life, Baltimore, Md.
H. Clive McAlister, M. D.	Lincoln National, Ft. Wayne, Ind.
Milton T. McCarty, M. D.	Peoples Life, Frankfort, Ind.
George McCreight, M. D.	Bankers Life, Des Moines, Iowa.
Francis H. McCrudden, M. D.	New England Mutual, Boston, Mass.
Carleton B. McCulloch, M. D.	State Life, Indianapolis, Ind.
Arthur J. McGanity, M. D.	Dominion Life, Waterloo, Canada.
Lewis F. MacKenzie, M. D.	Prudential, Newark, N. J.

H. Easton McMahon, M. D.	Mutual Life, New York City.
Walter T. McNaughton, M. D.	Old Line, Milwaukee, Wis.
Frederick W. McSorley, M. D.	Equitable Life Assurance, New York City.
Charles Maertz, M. D.	Union Central, Cincinnati, Ohio.
Henry L. Mann, M. D.	Equitable Life Assurance, New York City.
Robert W. Mann, M. D.	Imperial Life, Toronto, Canada.
William L. Mann, M. D.	Great West, Winnipeg, Canada.
Oscar F. Maxon, M. D.	Franklin Life, Springfield, Ill.
Samuel W. Means, M. D.	Metropolitan Life, New York City.
William H. Miller, M. D.	Equitable Life Assurance, New York City.
James T. Montgomery, M. D.	Southland Life, Dallas, Texas.
Richard C. Montgomery, M. D.	Manufacturers Life, Toronto, Canada.
John F. Moore, Jr., M. D.	Mutual Life, New York City.
William Muhlberg, M. D.	Union Central, Cincinnati, Ohio.
Alvin E. Murphy, M. D.	Mutual Life, New York City.
Charles T. Necker, M. D.	Dominion Life, Waterloo, Canada.
Charles F. Nichols, M. D.	Penn Mutual, Philadelphia, Pa.
John B. Nichols, M. D.	Acacia Mutual, Washington, D. C.
E. Clark Noble, M. D.	National Life, Toronto, Canada.

James H. North, M. D.	New York Life, New York City.
Ralph B. Ober, M. D.	Massachusetts Mutual, Springfield, Mass.
Herbert Old, M. D.	Provident Mutual, Philadelphia, Pa.
Martin I. Olsen, M. D.	Central Life, Des Moines, Iowa.
George P. Paul, M. D.	Aetna Life, Hartford, Conn.
William O. Pauli, M. D.	Union Central, Cincinnati, Ohio.
Charles B. Piper, M. D.	Connecticut Mutual, Hartford, Conn.
Joseph E. Pollard, M. D.	Prudential, Newark, N. J.
Roscoe W. Pratt, M. D.	New York Life, New York City.
William O. Purdy, M. D.	Equitable Life, Des Moines, Iowa.
William W. Quinlan, M. D.	Mutual Life, New York City.
James H. Ready, M. D.	General American, St. Louis, Mo.
William P. Reed, M. D.	Metropolitan Life, New York City.
Paul V. Reinartz, M. D.	Prudential, Newark, N. J.
Walter A. Reiter, M. D.	Mutual Benefit, Newark, N. J.
Frank P. Righter, M. D.	Atlantic Life, Richmond, Va.
George P. Robb, M. D.	Metropolitan Life, New York City.
James A. Roberts, M. D.	Canada Life, Toronto, Canada. Canada.

William C. Roberts, M. D.	New York Life, New York City.
A. J. Robinson, M. D.	Connecticut General, Hartford, Conn.
Oscar H. Rogers, M. D.	New York Life, New York City.
Henry B. Rollins, M. D.	Connecticut Mutual, Hartford, Conn.
Fred W. Rolph, M. D.	Confederation Life, Toronto, Canada.
Gordon Ross, M. D.	Massachusetts Mutual, Springfield, Mass.
Robert L. Rowley, M. D.	Phœnix Mutual, Hartford, Conn.
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